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SMOKING AND SOLDIER PERFORMANCE:

A Literature Review

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20. Abstract:

Research was reviewed on smoking as it relates to soldier performance for the US Army Medical Research and Development Command. This literature review resulted from an unsolicited proposal submitted by Research Solutions, Incorporated, in response to the Broad Agency Announcement of the Command. Research on smoking and other nicotine effects was included in the review. The research reviewed was related to position disclosure in combat; the effects of smoking on physical work capacity and endurance; the effects of smoking on perceptual processes; the effects of smoking on arousal and ability to deal with stress, pain, and fear; smoking-induced hormonal changes; the effects of tobacco deprivation; smoking-disease relationships and their effects on productivity and absenteeism; smoking and abuse of other substances, delinquency, and accidents; and associations between smoking and other factors of potential relevance to soldier performance. Among the main findings, the review disclosed detrimental effects of smoking on physical performance of soldiers, particularly soldiers with several years of tobacco exposure. The review also identified nicotine-related improved performance on vigilance and rapid information processing tasks, including tasks that may be relevant to some soldier tasks. It also showed a constellation of negative behaviors that are correlated with smoking such as drug abuse, delinquency, and driving accidents. Research in many areas critical to soldier performance, such as the effects of smoking on dark adaptation and the effects of smoking on testosterone production, showed contradictory results that need additional research for resolution. Needs for additional research on smoking and soldier performance were included as a final chapter of this report.

Table of contents

Introduction

Wars have been a large factor in the spread of tobacco use throughout the world and also in the increase of tobacco smoking and other tobacco use where it was already established (Van Proosdy 1960). The cigarette was a particularly convenient way to use tobacco in combat situations, and great increases in tobacco consumption occurred during the Crimean War and later during World War I (Ashton and Stepney 1982). These wars and World War II also sharply increased smoking in the civilian population "... due partly to a desire for an antidote to the heightened stresses and fatigue, ..." (Van Proosdy). More recently, Ben-Meir (1977) reported a survey of smoking habits after the 1973 Yom Kippur War that showed the first increase in smoking rates in the Israeli population for 3 yr. Israeli smokers also significantly increased the number of cigarettes smoked. Participants in two smoking cessation programs, who had shown sharp decreases in smoking, returned almost to preprogram smoking levels after the Yom Kippur War broke out. A subsequent program with these same participants failed to achieve earlier amounts of smoking reduction.

As a general rule, military personnel are much more likely to smoke than the general population of the country (John 1977, O'Malley, Bachman, and Johnston 1978, Van Proosdy 1960). A recently published study indicated that in 1977 smoking was higher for both male and female Air Force personnel than for the rest of the US population (Wetzler and Cruess 1985). Newspaper reports of current discussions of smoking in the military and of the sale of cigarettes in commissaries indicate soldiers still are more likely to smoke than their civilian counterparts. However, these specific survey results were not obtained for this review.

Smoking by US military personnel was actively encouraged for years when cigarettes were included with field-ration packets. Although cigarettes are no longer provided in field rations, military personnel still are encouraged to smoke by such things as the relatively low price of cigarettes in military retail outlets and regular breaks in military training activities that, at least some years ago, were frequently preceded by the announcement, "Smoke them if you have them."

Benefits and risks of smoking for soldiers and military operations

Reduction of stress is a major reason many people give for smoking (see Chapter 6: "Effects of smoking on arousal and

ability to deal with stress, pain, and fear"). Marshall (1947) reported many soldiers frequently were unable to function due to overarousal and fear during their first introduction to combat. Any reliable technique for calming the emotions might have major payoffs for performance of soldiers in combat situations. Of course, for many combat tasks such as those faced by dismounted infantry, smoking is probably impossible. Smoking is more compatible with soldier activities in armor, artillery, and support operations. However, even for the infantryman, breaks in the action or periods prior to the action, would allow time for smoking. No reports were found which indicated whether smoking or smoking deprivation affected the combat performance of soldiers who smoke, or whether they were more or less able to perform combat tasks than nonsmoking soldiers. Interviews with combat veterans could help meet this important research need (see Chapter 12: "Needs for additional research on smoking and soldier performance").

Although smoking is subjectively viewed as calming, smoking is almost always associated with an increase in physiological arousal as measured by heart rate, blood pressure, and changes in brain electrical activity (Gilbert 1979, Gilbert and Hagen 1980). This perceived calming of the emotions in the face of increased physiological arousal is known as Nesbitt's Paradox (Gilbert 1979, Schachter 1973). Despite much recent research on Nesbitt's Paradox, which will be reviewed in this report, it still is somewhat of a paradox. However, as will be described, some physiological processes such as sweating of the hands and muscular reflexes usually decrease with smoking, and for these processes there is no paradox. In addition, the physiological arousal with smoking has been shown to be relatively small compared to that resulting from other stressors like exercise or even the ingestion of fats (Sedgwick et al. 1981).

Research to be reviewed in this paper suggests that even in nonstressful situations, smoking may improve concentration in the face of distraction on cognitive tasks that are not unlike military tasks, such as computing artillery or mortar fire settings or monitoring and responding to a cathode-ray-tube (CRT) display of an air defense system such as Patriot. Such benefits from smoking would be expected to accrue particularly to the habitual smoker who would suffer additional distractions from withdrawal symptoms if he could not smoke (see Chapter 8: "The effects of tobacco deprivation"). On the other hand, much research reviewed in this report shows smoking has deleterious effects on physical work capacity, on health, and even possibly on very difficult problem-solving tasks (e.g., Elgerot 1976). These factors would operate to reduce soldier effectiveness, even if smoking did provide a bona fide reduction of combat stress or improvement in attention. Recent

research also has shown smoking reduces aggressiveness (Cherek 1981) and this might detract from performance in combat, which is the ultimate in aggressive behavior.

A major purpose of this review is to explore and weigh the benefits and liabilities associated with smoking by military personnel for performance of the Army's combat mission. This is no simple task. Smoking has a large number of different physiological and psychological effects including those mentioned above which should improve soldier performance and military operations. On the other hand, the effects of smoking on endurance and health appear to be primarily bad. Smoking also discloses soldier positions, starts fires, and contributes to the cause of vehicular accidents. Smokers also are much more likely than nonsmokers to use other drugs and to get into trouble.

Despite thousands of research studies on smoking and its effects on human beings, only a small fraction of these studies have been specifically directed at soldier performance or military operations. As a result, this paper will frequently describe applied research needed to determine whether "established" effects of smoking on behavior really are applicable to military activities which often are unique in their character and intensity. Policy makers reading this report undoubtedly will be frustrated by these frequent calls for more research before a beneficial or detrimental effect of smoking can be established in various general and specific military contexts. However, the research proposed is straightforward and results could be obtained in a few months following initiation of these projects.

Overview of this report

This review will cover the following topics: position disclosure in combat due to smoking and tobacco-seeking behavior; effects of smoking on physical work capacity and endurance; effects of smoking on vision and perceptual processes; effects of smoking on vigilance, rapid information processing, and divided attention; effects of smoking on cognitive processes; effects of smoking on arousal and on the ability to deal with stress, pain, and fear; smoking-induced hormonal changes; the effects of tobacco deprivation; smoking-disease relationships and their effects on productivity and absenteeism; the relationship of smoking to abuse of other substances and to other negative traits and behaviors; associations between smoking and other factors of potential relevance to soldier performance; and needs for additional research on smoking and soldier performance. For the most part, chapters of this report correspond to each of the above topics. In

those chapters where the data are primarily "psychological" or related to physical performance, much more description, discussion, and evaluation of results occur than in chapters where primarily physiological and medical research is reviewed. This reflects the background, experience, and training of the author who is an experimental psychologist and a marathon runner with many years of research on performance of infantry soldiers and their leaders.

The report will not discuss research on why people start to smoke, nicotine regulation, or techniques for reducing smoking. This is not because these topics are not important to the Army or other military branches, but because they are less directly relevant to the topic of smoking and soldier performance, and because available resources limit the scope of this report. For the reader interested in research on nicotine regulation, i.e., the tendency for the habitual smoker to maintain constant levels of nicotine despite different intervals between cigarettes or different nicotine levels of cigarettes, this subject has recently been critically reviewed by McMorrow and Foxx (1983). A brief recent review of smoking cessation programs and other attempts to control smoking has been provided by Fielding (1985b)

Intended audiences for this report

The intended audiences for this report include research scientists who study soldier performance, senior Army leaders and policy makers, and commanders at the battalion level and above (and their staffs). Policy makers of the Army (and other services) need the information provided in the report related to 1) the possible benefits associated with smoking for stress management, performance of vigilance tasks, performance of rapid information processing tasks, etc.; 2) the confirmed large and unambiguous dangers smoking presents to soldier health, physical performance, and combat position disclosure; 3) the possible problems associated with depriving habitual smokers of tobacco; and 4) the constellation of negative behaviors (e.g., delinquency, alcoholism, and drug abuse) that frequently are associated with tobacco use.

Policy makers might wish to consider tobacco use as a factor in selection of military personnel. It is probable, given strong influences on smoking of peers (e.g., Antonuccio and Lichtenstein 1980), older siblings (Spielberger et al. 1983), and teachers (Murray, Kiryluk, and Swan 1984), that smoking is higher in units where the leader smokes, although this probable relationship of the smoking-status of leaders to rates of smoking of their soldiers also has not been the subject of research (see Chapter 12: "Needs for additional

research on smoking and soldier performance"). If nonsmoking personnel were available and other relevant factors were equal, selection of nonsmokers for leadership positions might be a useful strategy for reducing smoking in the Army.

Policy makers also might consider other forms of nicotine administration as a means to improve performance or to replace smoking. Although carbon monoxide and its associated blood compound carboxyhemoglobin have been shown to be associated with atherosclerosis, endothelial cell damage, and arterial lesions,[1] and although other components in cigarette smoke such as benzopyrene and polonium, have been implicated in development of lung cancer (Clee and Clark 1982), nicotine alone may not be particularly dangerous to health, when taken in smoking-sized-doses through other means than smoking or other forms of tobacco use. If the benefits of nicotine for performance on vigilance and rapid information processing tasks (e.g., Wesnes 1985) and the apparent benefits of nicotine for stress reduction (e.g., Nesbitt 1973) are to be exploited in combat situations, then it should be through a form of nicotine administration such as pills, chewing gum, or nasal sprays (Russell et al. 1983, West et al. 1984a), instead of through inhalation of the dangerous fumes produced by burning tobacco products. However, more research is needed on these "benefits" of nicotine, as well as research on the health, addiction, and other possible consequences of these new forms of nicotine administration before they should be considered by policy makers for Army use.

Army leaders at all levels should become aware of the problems that soldiers who smoke pose to unit position disclosure in combat, of the performance decrements that can be expected in "addicted" smokers in NOPF environments and other situations where smoking is impossible, and of the general decrements in physical endurance, and to a lesser extent sensory performance, associated with breathing carbon monoxide and other components of tobacco smoke.

Research scientists who study performance of military personnel are the third main audience for this report. As Tong et al. (1974b) pointed out for behavioral scientists in general, military psychologists and other scientists who investigate soldier performance must become aware of the effects tobacco use has on almost every facet of human behavior. The effect of smoking on performance during intense stress, such as that associated with combat (or even during the lesser stress of airborne training), has not been studied and

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[1] Allen, Kluft, and Brommer (1985) have provided a brief review of health hazards of low COHb levels.

this appears to be a gross oversight given the large payoffs from smoking that the habitual smoker, who smokes to reduce stress, would undoubtedly predict. Research also is needed to determine whether the sharp increase in muscle tremor produced by tobacco use causes decrements in rifle marksmanship and marksmanship with other hand-held or hand-guided weapons. Smokers who routinely inhale through tiny filters may actually perform better while wearing the protective (gas) mask, at least until nicotine-withdrawal symptoms appear, and it may be no coincidence that young male smokers sometimes have been shown to have better lung function than their nonsmoking peers (Tashkin et al. 1983). Research is needed to determine if these and other important military tasks are impaired or facilitated by smoking. Vigilance task performance, which typically is enhanced by smoking (e.g., Wesnes and Warburton 1978), needs further investigation with bona fide military vigilance tasks. Research is needed to see if benefits of nicotine and smoking for laboratory "rapid information processing" tasks presented on CRTs also apply to the similar rapid information processing tasks operators of weapons systems such as Patriot and Aquila will perform in combat settings. These issues (and many others discussed throughout the report, especially, in Chapter 12: "Needs for additional research on smoking and soldier performance") need additional research and evaluation in field environments to provide information to assist the policy makers and leaders.

Much of this report consists of technical material which will be of primary interest to scientists investigating soldier performance, smoking, or both, although this is not true for the next brief chapter on smoking and position disclosure in combat where the only material located was largely anecdotal. At the end of each major chapter, conclusions will be presented along with military implications (if sufficient knowledge has been gained to have implications). Commanders and policy makers may wish to concentrate on these Conclusions and military implications sections of the major chapters.

Researcher and reviewer biases

One can guess in many research reports on smoking whether the researcher is a smoker or a nonsmoker. Sometimes small differences favoring nonsmokers are described as large when the result might lead policy makers to restrict smoking (e.g., Robinson and Wolfe 1976). The opposite tendency may be somewhat less prevalent, but it also exists (e.g., Dille and Linder 1981). A recent book (Tollison 1986) is aimed at achieving "a more balanced assessment." However, it is interesting that emphysema is not even mentioned in the book, including the chapter by Eysenck on smoking and health (that

questions the smoking-lung-cancer link), and this author would question the book's "balance." Hopefully, this exsmoker has prevented his prejudices against smoking from influencing selection, presentation, and discussion of research, and this report is a fairly objective account of the research literature on smoking and its implications for soldier performance.

Chapter 1

Position disclosure in combat due to smoking and tobacco-seeking behavior

Most of the literature found on position disclosure due to smoking was of an anecdotal nature. For example, a 1944 Bill Mauldin cartoon shows a medic lighting his pipe at night to the great concern of some nearby troops. "It's okay, Joe. I'm a noncombatant," is the caption (Mauldin 1968).

The superstition, "Three on a match is bad luck," apparently originated during the Boer War when British soldiers, who were the third person to have their cigarettes lighted with the same match, became frequent casualties of Boer President Kruger's snipers. "It was argued that the sniper saw the flame as the first cigarette was lighted, took aim at the illumination for the second cigarette and fired when the third light was being given (Radford and Radford 1949)."

A review of military literature pertaining to combat in this nation's wars found some discussion of "light discipline" with the major light discipline problems being associated with lighting and smoking cigarettes (Bussey 1965).

"Combat Tips for Fifth Army Infantry Replacements in Italy," (US Army 1945) included the following warnings:

> "If you smoke or make a fire at night, be sure the glow cannot be seen from any direction. Don't smoke on guard because the glow of a cigarette can be seen for a long way."
>
> "Smoking is another thing to be careful about. In the words of a captain, 'Too many men are careless about cigarettes when they think they are far from Jerry. All we shoot at after dark is lights, and it is the same with Jerry.' Don't smoke at night out of doors. Get in your dugout or under a shelter half."

Light-amplification devices and infrared telescopes presumably make the avoidance of the lighting and smoking of cigarettes in combat situations even more important today (Infantry staff 1977).

No references to tobacco-seeking behavior and combat casualties were located. However, given the persistent drive for tobacco in the habitual user (e.g., Schachter 1978), it is reasonable to assume unnecessary exposure to enemy fire

occurred in order to satisfy a need for tobacco, with frequent tragic consequences.

Given the legends, cartoons, and other material dealing with the subject, the general absence of literature relating smoking and tobacco-seeking to position disclosure was somewhat surprising. This may reflect the inappropriateness of some research activity in combat situations. It also may reflect the impossibility for many victims and an unwillingness of survivors to document this "trivial" basis for casualties. On the other hand, combat veterans from every war since 1917 probably could provide important information on this subject based on their combat experience. Research is needed in which these men are carefully interviewed to determine the extent of problems associated with smoking and tobacco seeking for position disclosure. Such veterans simultaneously could provide other important information related to the effects of smoking on combat performance such as the possible enhancement of performance of new troops through stress reduction (see Chapter 12: "Needs for additional research on smoking and soldier performance").

Chapter 2

Effects of smoking on physical work capacity and endurance

Interviews with active duty and retired infantry personnel with combat experience indicate physical endurance and strength are even more important for successful performance in combat than they are for successful performance on the athletic field. During the smoking of tobacco (or other substances), the inhaled products of combustion would be expected to reduce a person's capacity for work if for no other reason than the fact that these products dilute the oxygen in his lungs. However, one major component of tobacco smoke is carbon monoxide (CO), which does much more than dilute or displace the oxygen in the lungs. It combines with the oxygen-transmitting hemoglobin of the blood forming carboxyhemoglobin (COHb). This reduces the amount of hemoglobin available to transport oxygen from the lungs to the tissues (Castleden and Cole 1975). COHb also increases the oxygen affinity of the remaining oxyhemoglobin, so that oxygen is given up to the tissues less readily (Roughton and Darling 1944).[1]

According to the National Academy of Sciences (1977), cigarette smoke is the major source of CO in indoor environments (vehicular exhaust being the major source of CO outdoors). Although estimates of CO in tobacco smoke vary widely (Aviado 1984), both for mainstream (puffed) smoke and sidestream smoke (smoke as it comes directly from an unpuffed cigarette), the level of CO in smoke as it comes directly from an unpuffed cigarette apparently can be as much as five percent (Castleden and Cole 1975). In tobacco smoke that is inhaled, CO is present at levels of 400 to 500 ppm (US Department of Health Education and Welfare 1979). Goldsmith, Terzaghi, and Hackney (1963) estimated the cigarette smoker is exposed to 475 ppm CO for approximately 6 min for each cigarette smoked. This is much higher than the Environmental Protection Agency maximum concentration of CO for a 1-h exposure which is only 35 ppm, and for an 8-h exposure which is only 9 ppm (General Services Administration 1984).

When CO is breathed in the environment or in cigarette smoke, the COHb level in the blood increases rapidly (National Academy of Sciences 1977). Castleden and Cole (1975) found the level of COHb in the blood of smokers averaged 3.8 percent for persons smoking 1 to 10 cigarettes daily, 6.1 percent for

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[1] However, Vogel et al. (1972) failed to find any additional effect of COHb other than the proportionate reduction of oxygen transport in their research on CO effects on performance.

persons smoking 11-20 cigarettes daily and 6.7 percent for persons smoking more than 20 cigarettes daily. However, inhalation patterns strongly influence COHb levels and some heavy smokers have been found to have COHb levels of ten percent (Seppanen 1977) and even above ten percent (Russell, Cole, and Brown 1973). Levels in nonsmokers averaged .68 percent in one study (Raven et al. 1974b), 1.3 percent in another (Castleden and Cole 1975) and about 1.8 percent in yet another (Seppanen 1977). These nonsmoker levels of COHb vary depending on the ambient levels of CO. Levels of COHb as high as 4 percent have been measured in nonsmokers who were subjected to an environment where extremely heavy smoking occurred (Russell, Cole, and Brown 1973).

Effect of carbon monoxide and smoking on the physiological response to exercise

Levels of COHb well above those resulting from smoking (20 percent or greater) often produce dramatic reductions in physical and mental performance, and a COHb level of 67 percent generally results in death if untreated (Stewart 1975). Vogel et al. (1972) produced COHb levels of 20 percent, and found a 20 percent reduction in maximum oxygen utilization (VO_2 Max) during performance on a bicycle ergometer. Pirnay et al. (1971) produced COHb levels of 15 percent and found a corresponding 15 percent reduction in maximum oxygen utilization during performance on a treadmill. Both studies indicated CO exposure diminishes work in proportion to the level of COHb of the blood. Horvath et al. (1975) reviewed studies that measured performance with different levels of COHb and provided an equation that described this nearly linear relationship of the change in maximum performance to level of COHb (Percent Change of VO_2 MAX = 0.91 X Percent COHb + 2.2).

Given this approximately equal reduction of work capacity and of the oxygen-carrying capacity of the blood, it is not surprising that there is little change in physical performance as a result of increasing COHb to the relatively low levels produced by smoking when the levels are achieved by breathing mixtures of CO and air. Some researchers have found no performance decrements with these small increases in COHb levels. For example, in a study where 50 ppm CO-air mixtures were inhaled to increase COHb levels, Raven et al. (1974b) did not find decreased performance over baseline levels in young smokers and nonsmokers whose average age was 24.5 years. COHb rose to 2.7 percent from .64 percent for nonsmokers and rose to 4.5 percent from 3.2 percent for smokers. Another study of middle-aged men (average age = 47.5 years) involving the same CO-air treatment (Raven et al. 1974a), showed differences between smokers and nonsmokers (see below), but, as in Raven et

al. (1974b), the small increases in COHb from breathing 50 ppm CO did not reduce performance compared to baseline performance of either the smokers or nonsmokers. However, it should be noted these levels of COHb are somewhat lower than those for moderate to heavy smokers (Castleden and Cole 1975).

A number of other studies have found significant reductions in performance on treadmill or bicycle ergometers at the levels of COHb produced by smoking. Seppanen (1977) found COHb levels in the ten percent range which resulted from breathing CO-air mixtures produced significant decrements in maximal work level measured on a bicycle ergometer. Ekblom and Huot (1972) used laboratory measures of physical work done on treadmills and bicycle ergometers and showed a reliable decrease in physical work capacity as a result of blood COHb levels as low as 4.8 percent. Horvath et al. (1975) found 4.3 percent COHb to be the lowest level in their study to produce a significant decrement in performance.

Aronow and Cassidy (1975) used a double-blind crossover design and found a five percent decrease in time to exhaustion following CO breathing (100 ppm CO) compared to time to exhaustion while breathing compressed, purified air. COHB increased to 3.95 percent from 1.67 percent following CO breathing and decreased to 1.30 percent from 1.63 percent following breathing of compressed air. They concluded COHb levels of the magnitude produced by cigarette smoking significantly reduced performance with the mechanism probably being impairment of myocardial oxygen delivery.

Aronow and Cassidy (1975) did not report whether or not VO_2 Max differed between CO-breathing and air-breathing conditions. Presumably, it did not, even though time to exhaustion did. This result is typical of several studies that have shown the time to exhaustion in progressive tests of maximal oxygen utilization is significantly reduced following induction of smoking-level levels of COHb while VO_2 Max has not shown a significant difference. The implication is that measures of VO_2 Max are less reliable and less valid measures of performance than is the time on a treadmill or a bicycle ergometer until exhaustion is reached.

Exposure to 50 ppm CO "significantly" reduced treadmill walking time for nonsmokers, but not for smokers in a study by Drinkwater et al. (1974). VO_2 Max did not show significant differences as a result of CO exposure for either group. COHb levels only increased to 2.5 percent from .9 percent in nonsmokers and to 4.1 percent from 2.6 percent in smokers. However, this result may be an artifact since the variance for walk time for nonsmokers in the CO condition is about double that for nonsmokers in the other conditions in the experiment.

Although CO appears to be the major factor in tobacco smoke that reduces capacity for work, the question exists whether COHb levels produced by smoking have the same effect on performance as comparable COHb levels produced by breathing CO from nontobacco-smoke sources. In other words, does nicotine or does some other tobacco smoke component beside CO influence performance?[2] The study by Seppanen (1977) compared work capacity for equivalent COHb levels (10 percent) when the levels were produced either through breathing 1,100 ppm CO from an air bag or through smoking. These levels of COHb are high for smokers and for studies of "smoking-level" COHb effects. For a given submaximal heart rate, the level of work with elevated COHb was significantly less than the level obtained while breathing normal air. This effect held for three intermediate levels of work measured by heart rates of 130-, 150-, and 170-beats-per-minute. Seppanen also found the smoking condition caused a larger decrease in performance for a given submaximal heart rate from performance while breathing ordinary air than the comparable decrease in performance that was caused by 1,100 ppm CO inhalation from an air bag. Presumably, increased work of respiration due to bronchoconstriction (Klausen, Andersen, and Nandrup 1983), reduced venous return (Krone et al. 1972), reduced heart stroke volume (Goldbarg, Krone, and Resnekov 1971), or some factor associated with components of cigarette smoke, other than the CO, reduced the capacity for pedaling the bicycle ergometer.

Seppanen (1977) found the maximum level of work under both conditions of elevated COHb also was significantly less than the maximum level obtained in the condition of breathing of normal air. However, the finding of worse performance with smoking-based COHb than CO-air-based COHb at the three submaximal heart rates was reversed during the assessment of maximal work level. Greater maximum work levels resulted on the bicycle ergometer when tobacco smoke produced the COHb than when it was produced by breathing a CO-air mixture. However, this difference between CO-air-mixture inhalation and tobacco-smoke inhalation did not reach statistical significance and this superiority of the tobacco-smoking condition probably is an artifact. Both the lack of statistical significance and the lower average performance probably are related to the fact that the variance of the measure of maximal work for the CO-inhalation condition (3,249) was about four times that of the air-breathing condition (900) and tobacco-smoking condition (729). No such inflation of variance occurred for the CO-inhalation condition at any of the lower heart rates. A

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[2] Tobacco smoke contains thousands of distinct tobacco products with the most significant active constituents being tar, carbon monoxide, and nicotine (Henningfield 1984).

ceiling effect on performance scores would exist in this maximum work performance condition and this implies the scores which inflated the variance during CO-inhalation were, for the most part, lower, not higher than the average. Any attempt to explain this lower average level of maximal work for CO-inhalation than for tobacco-smoke inhalation should account for the unusually low scores for CO-inhalation rather than through explanation of the "elevated" scores for the tobacco-smoke inhalation condition.

Despite this inflated variance for performance following CO-inhalation and despite the lack of significance of the difference between CO inhalation and tobacco-smoke inhalation, Seppanen (1977) discusses the higher level of performance following smoking as if it were significant and he proposes a tentative explanation of these "elevated" scores for the smoking condition. According to Seppanen, the nicotine from tobacco smoking apparently acted as a stimulant during the exhaustive work, overcoming some of the deleterious effects of COHb.

If Seppanen (1977) were correct, such a stimulating effect of tobacco also might account for the results of several studies that have shown little or no difference between VO_2 Max of smokers and nonsmokers (e.g., Chevalier et al. 1963, Krumholz, Chevalier, and Ross 1964) and between smoking and nonsmoking subjects (e.g., Raven et al. 1974b). Heart rates found in these studies were typically higher in smokers than nonsmokers during the exercise period and nicotine-boosted cardiac output may be the explanation for the smokers' ability to perform as well as nonsmokers on these tasks, despite the COHb-reduced oxygen-carrying-capacity of the smoker's blood.

Effects of exercise on carboxyhemoglobin levels

Many studies of COHb effects have discontinued CO breathing during the period of actual exercise while others (e.g., Pirnay et al. 1971, Vogel and Gleser 1972) have maintained continuous breathing of CO-air mixtures during the exercise period. At least two studies that discontinued CO breathing showed striking COHb clearing effects of maximal exercise testing. Klausen, Andersen, and Nandrup (1983) reported a drop during about 8 min of testing to 3.39 percent COHb from 5.26 percent COHb for CO-breathers, a drop to 2.59 percent from 4.51 percent for smokers, and even a drop to 0.86 percent from 1.51 percent for control subjects who did not smoke or breath CO prior to testing. Hirsch et al. (1985) made a similar finding with COHb dropping to .9 percent from 1.8 percent during the nonsmoking test and to 4.8 percent from 6.6 percent during the smoking test. Test durations were approximately 11 min. Given

that elevated COHb levels may influence mental as well as physical functioning (e.g., Stewart 1975), brief periods of cardiovascular exercise could have important beneficial effects on both forms of performance, especially for smokers who show larger absolute (though smaller percentage) changes.

Smoker-nonsmoker differences when exercise testing is not immediately preceded by smoking

Several studies have shown little or no difference between smokers and nonsmokers on VO_2 Max (Chevalier et al. 1963, Krumholz, Chevalier, and Ross 1964, Maksud and Baron 1980). A possible explanation of the absence of differences in VO_2 Max between young smokers and nonsmokers is that lung function is higher, on the average, for boys who take up smoking than for boys who do not and it is only after several years of smoking that they become equal and with continued smoking eventually become worse (Tashkin et al. 1983).

Raven et al. (1974b) also found no differences in maximum work output (VO_2 Max) between young smokers and nonsmokers (average age = 24.5 yr) either before or after breathing CO-air mixtures. However, another study of middle-aged men (average age = 47.5 yr) from the same laboratory (Raven et al. 1974a) used basically the same procedures and same CO-air treatment and showed large differences between smokers and nonsmokers. As in Raven et al. (1974b), low levels of COHb produced by breathing CO-air mixtures did not change baseline performance of either the smokers or the nonsmokers. However, large and significant differences did appear between these middle-aged smokers and middle-aged nonsmokers in VO_2 Max both at baseline and after breathing CO-air mixtures. This occurred despite the fact that prescreening of smokers already had led to one of every two candidates who smoked being rejected because of abnormal exercise electrocardiograms. Raven et al. (1974a) compared the smoker-nonsmoker difference for the younger men of Raven et al. (1974b) with the smoker-nonsmoker difference for middle-aged men of Raven et al. (1974a). This comparison indicated increased age was associated with only a six percent drop in VO_2 Max for nonsmokers, but a 26 percent drop in VO_2 Max for the smokers. Smokers averaged 4.3 yr older than the nonsmokers in the middle-aged group and some small part of this effect may be related to this age difference. However, the large difference in VO_2 Max between these older smokers and older nonsmokers surely would have held even had average ages been equivalent. It would appear from Raven's reports that smoking produces large decrements in performance, but these take several years of smoking to make their appearance. Support for this also comes from a cross-sectional study by McHenry et al. (1977) who showed no difference between

nonsmokers and current smokers in the maximal duration of exercise when the current smokers had ten or less pack-yr of smoking exposure.[3] For smokers with more than ten pack-yr of smoking exposure, duration of exercise was significantly shorter (889 s versus 958 s). Age differences between the different smoking exposure group were small and average age was actually less for the greater-than-ten-pack-yr exposure group than for the nonsmokers.

Keith and Driskell (1982) compared the VO_2 Max of 12 smokers and ten nonsmokers who ranged in age from 25 to 38 yr in a study looking at the effects of Vitamin C on athletic performance. Although VO_2 Max was higher for nonsmokers (19.6 ml/kg/min) than for smokers (17.3 ml/kg/min), the differences did not reach statistical significance. A 3-wk regimen of 300 mg of ascorbic acid daily did not increase performance in either group.

Unlike the studies that typically showed no smoker-nonsmoker differences in performance for young subjects, Montoye, Gayle, and Higgins (1980) examined performance for subjects of different ages and found their largest smoker-nonsmoker VO_2 Max differences to occur for 16-24 yr-old men. These results are in sharp contrast to the results of the studies of Chevalier et al. (1963), Krumholz, Chevalier, and Ross (1964), and Raven et al. (1974b) which did not find differences in their young subjects. In the study of Montoye, Gayle, and Higgins, measurements were made during walking performance on a treadmill instead of during bicycle ergometer pedaling, and the test was much longer. For younger males, the test required a walk to exhaustion with an "adjusted" mean time on treadmill being over 24 min for nonsmokers and less than 22 min for smokers. However, it is not clear why the difference between the testing procedure of Montoye, Gayle, and Higgins and those of Chevalier et al. and Krumholz, Chevalier, and Ross should produce these atypical striking differences between young smokers and young n nsmokers for Montoye, Gayle, and Higgins.

Montoye, Gayle, and Higgins (1980) also reported a lack of difference between their older smokers and nonsmokers. This was in strong contrast to Raven et al. (1974a) who found a striking difference between older smokers and nonsmokers. Montoye, Gayle, and Higgins attributed this to the elimination of many older nonsmokers prior to the study because of health problems that prevented treadmill testing. However, Raven et al. (1974a) also eliminated many older subjects for this reason

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[3] A pack-yr of exposure is defined as the product of the number of packs per d times the number of yr of smoking.

in their research. Although some small differences existed between the testing procedures of Raven et al. and Montoye, Gayle, and Higgins, it is difficult to account for the large difference in performance between older smokers and older nonsmokers for the former and no such difference for the latter.

It should be mentioned, however, that the surprising difference between young smokers and young nonsmokers and the surprising absence of difference between older smokers and older nonsmokers are not the only unusual findings in the Montoye, Gayle, and Higgins research. They also reported nondrinkers performed significantly worse on treadmill tests of VO_2 Max than the drinking group, despite the fact that 1) 85 percent of drinkers were smokers, 2) only 47 percent of nondrinkers were smokers, and 3) smokers performed significantly worse than nonsmokers. It is probable that at least some of the covariates (these included age, weight, skinfolds, smoking habits, and drinking habits) were inappropriate because their relationship to performance was nonlinear or for other reasons. Thus, inappropriately analysis-of-covariance-adjusted averages may account for many of these anomalous results.

Immediate effects of smoking on physical performance

Hirsch et al. (1985) compared maximal bicycle ergometer performance of nine young smokers (average age 24.4 yr) on smoking days and nonsmoking days and found that VO_2 Max was reduced significantly on smoking days compared to nonsmoking days. They concluded cigarette smoking causes immediate detrimental effects on cardiovascular function during exercise, including increased heart rate, a lowered anaerobic threshold, and impaired oxygen delivery to muscles. Carbon monoxide and nicotine were implicated in these changes to a greater extent than the effects of smoke particulates since respiratory function was largely unchanged from smoking to nonsmoking days.

Although Hirsch et al. claimed to have provided the first study of the immediate effects of smoking, several other studies already had addressed this issue including the study by Seppanen (1977) which compared smoking-produced COHb with CO-produced COHb and had compared both conditions to a condition where COHb was reduced to nonsmoker levels by air breathing prior to testing. As described earlier, both conditions with elevated COHb produced significantly lower VO_2 Max than the air-breathing condition.

An even earlier study by Goldbarg, Krone, and Resnekov (1971) compared bicycle ergometer performance of young subjects

after abstention from smoking for at least 12 h and immediately after smoking one cigarette. Although oxygen consumption for a given work load increased after smoking, the change was not significant. On the other hand, heart rate increased and stroke volume decreased following smoking with these changes significant at all exercise levels.

In another repeated-measures study where performance of "moderate habitual" smokers was compared after 12 h of smoking deprivation, after inhalation of three cigarettes and after inhalation of CO, Klausen, Andersen, and Nandrup (1983) found both smoking and CO inhalation reduced VO_2 Max and work time on a bicycle ergometer relative to the control condition of 12 h of abstinence from smoking. Reduction in VO_2 Max from the smoking deprivation condition was about 10 percent for both CO-breathing and smoking. Work time was significantly less in the tobacco-smoke condition than in the CO condition, although COHb levels were comparable. Smoking produced 3.55 percent COHb and CO-breathing produced 3.94 percent COHb. Given the low levels of COHb, the formula of Horvath et al. (1975) would predict less change in performance than the 10 percent found in this study.

Klausen, Andersen, and Nandrup (1983) found maximum heart rate to be significantly lower in the smoking condition than in the control condition, while resting heart rate and heart rate during recovery from exercise were higher in the smoking condition. This reduced heart rate in the smoking condition during maximum exercise was not found by Seppanen (1977) in his similar repeated-measures research. However, Seppanen required much more smoking as is evidenced by COHb levels in the range of ten percent compared to less than four percent in the study by Klausen, Andersen, and Nandrup.

Other evidence of immediate detrimental smoking effects on performance was provided by Brundin (1980) who used light and moderate submaximal work conditions on a bicycle ergometer and looked at the effect of smoking a cigarette during the light exercise condition and of smoking two cigarettes during a 30-40 min intermission between two 8-min exercise sessions where the exercise was somewhat more intense. Subjects in a control group did not smoke during or between exercise sessions. Heart rate and blood temperature were elevated significantly as a result of the smoking and this was accompanied, and probably caused, by an increase in metabolism required to perform the tasks while smoking. Reduction of skin blood flow occurred during the task requiring low effort and this also may have contributed to the blood temperature increase for the smoking condition. Smoking during the higher effort condition did not result in reduced peripheral blood flow.

Morton and Holmik (1985) compared performance of 14 "well-trained team sportsmen" when the test of maximal oxygen consumption was preceded by smoking and on another day when it was not preceded by smoking. Half of these athletes were smokers and half were nonsmokers, but all smoked for the experiment. Morton and Holmik found smoking reduced VO_2 Max for both smokers and nonsmokers, but the differences were not significant. However, durations of the treadmill tests used to measure maximum performance were significantly shortened by 32 s (3.5 percent of the average duration of 14.28 min) following smoking for both smokers and nonsmokers with a post-hoc test indicating more shortening was found for the nonsmokers. Less fit persons than these "well-trained team sportsmen" probably would have shown larger differences in performance as a result of smoking given another result of Klausen, Andersen, and Nandrup (1983) who found their less fit subjects to show the greatest acute effect of smoking on maximum performance.

Morton and Holmik (1985) found no initial or post-smoking differences between smokers and nonsmokers in VO_2 Max. Age of these men was not given, but they were probably in their late teens, 20s, or early 30s. Older persons probably would have shown larger differences in performance between smokers and nonsmokers given the results of Raven et al. (1974a) and McHenry et al. (1977).

Ratings of perceived exertion were shown by Ekblom and Goldbarg (1971) to be sensitive to fitness differences (as indicated by heart rate differences during exercise). Morton and Holmik (1985) obtained such ratings once per min during treadmill testing. No differences appeared between smoking and nonsmoking groups in perceived exertion, but perceived exertion was rated significantly lower during periods of testing following the smoking of two cigarettes for both smokers and nonsmokers. This reduction in perceived exertion following smoking might reflect a change in pain tolerance with smoking such as reported by Nesbitt (1973) and more recently by Pomerleau, Turk, and Fertig (1984).[4] Such a change in perceived exertion conceivably could allow harder efforts following smoking and this might counter COHb or other smoking-related performance limiters. On the other hand, two studies have shown exercise at a given heart rate is perceived to be harder by smokers than nonsmokers (Hughes et al. 1984a, Maksud and Baron 1980), although this may reflect a long-term effect

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[4] These changes in pain tolerance with smoking are described in detail in Chapter 6: "Effects of smoking on arousal and ability to deal with stress, pain, and fear."

of smoking where the Morton and Holmik result is an immediate smoking effect.

Rode and Shephard (1971) showed abstention from smoking for a single d was associated with a 13-percent to 19-percent decrease in the amount of oxygen required to support muscles involved with breathing during near-maximal exercise. Pulmonary changes thus appear to be one mechanism for acute detrimental effects of cigarette smoking. Rode and Shephard reported their six subjects, who ranged in age from 24 to 46, all reported treadmill exercise was easier to perform on nonsmoking runs than on smoking runs. This appears to conflict with the results of Morton and Holmik who reported lower perceived exertion following smoking and the results of Myrsten, Elgerot, and Edgren (1977) who found no perceived exertion differences between smokers and abstainers. The effects of smoking on perceived exertion need replication and clarification in additional studies.

Myrsten, Elgerot, and Edgren found 5 d of abstinence from smoking reduced the heart rate required for bicycle exercise at a level of 150 watts by nearly nine beats per min compared to a group of subjects who did not abstain. However, perceived exertion for the task did not differ for the two groups. Abstainers and nonabstainers also were allowed to choose a preferred work level in this research. Smokers who abstained chose a higher work level after the 5-d abstention period than those who continued to smoke. Following another 5-d period when the abstainers resumed smoking, they still maintained a higher preferred work level than those smokers who had not abstained at all. If additional research were to show this effect of smoking abstention on preferred work level to be a reliable phenomenon, it would provide another reason for at least temporary abstention from smoking for soldiers.

Physical fitness test differences between smokers and non- smokers

Although the comparisons of smokers and nonsmokers on laboratory measures of physical performance have shown mixed results for young subjects, there is much more consistency in results from recent comparisons of smokers and nonsmokers on military physical fitness tests and other tests of physical fitness. These studies typically have shown average performance of smokers to be lower than the average performance of nonsmokers. For example, Cooper, Gey, and Bottenberg (1968) tested 419 airmen (average age 19.1 yr) on the maximum distance they could cover by running or by running and walking in 12 min. At least 1 h of smoking abstinence occurred prior to testing to reduce or eliminate the influence of acute smoking

effects. Amount of smoking, duration of smoking, and inhalation all significantly reduced the distance covered in 12 min. Forty-seven of these airmen also received treadmill tests of maximum performance. Although no significant difference in VO_2 Max was found between smokers and nonsmokers, maximum oxygen consumption did correlate strongly with 12-min run distances for both smokers (r=.69) and nonsmokers (r=.75). Other research by David (1968), Hartling (1975), and Kujala (1981), provided differences favoring nonsmokers over smokers similar to those obtained by Cooper, Gey, and Bottenberg. However, there is at least one exception. Pleasants (1969) found no difference in pretraining swimming tests between smokers and nonsmokers despite his expectation that decreased diffusion capacity and increased oxygen debt found in previous research would reduce significantly smoker performance and despite earlier research by Cureton (1936) who found nonsmokers to be superior to smokers. The subjects in the study of Pleasants were 18- to 20-yr-old males in intermediate college swimming classes and distances were 100 and 200 yd.

Recent research by Jensen (1986) has shown differences between smokers and nonsmokers also appear on the current Army Physical Readiness Test (APRT). He compared APRT performance of enlisted medical personnel in a medical company who were smokers, former smokers, and nonsmokers. Despite small numbers in each group, significant differences appeared between smokers and nonsmokers on the 2-mi-run test and the pushup test. Smoking males ran 13 percent slower than nonsmokers and females who smoked ran 8 percent slower than their nonsmoking counterparts. Males who smoked did 16 percent fewer situps and females who smoked did 18 percent fewer situps. Differences favored nonsmokers for the pushup event, but were not significant. Average age of these soldiers was 27.4 yr for males and 24.8 yr for females. Soldiers over 39 yr were not included because of different APRT grading procedures. Former smokers typically took intermediate values on performance measures between smokers and nonsmokers, but they numbered less than half the number in the other groups and differences were not significant.

Biersner, Gunderson, and Rahe (1972) studied naval personnel who volunteered for physically stressful Underwater Demolition Team Training (UDTT) and found smokers to perform significantly worse on a fitness test (squat-jumps, sit-ups, and pull-ups) than nonsmokers. They also found amount of smoking to be related to fitness. Nonsmokers performed significantly better on fitness tests than the group who smoked "some." Smokers who indicated they smoked "some" performed significantly better than smokers indicating they smoked "a lot."

These UDTT volunteers studied by Biersner, Gunderson, and Rahe appear to be a special group of the Navy enlisted population. They were only one-fourth as likely to smoke "a lot" (9.2 percent) as the general Navy enlisted population who smoked "a lot" (37.5 percent), and were nearly twice as likely not to smoke at all (51 percent vs. 26.5 percent). They will be discussed again in Chapter 10 which describes negative traits that frequently are associated with being a smoker, and positive traits that are frequently associated with not being a smoker. It is probable at least some of the consistent differences in physical fitness test performance between smokers and nonsmokers may reflect motivational differences or other personality differences that lead to increased effort by nonsmokers. Such increased effort during physical training would in turn lead to increased physical capacity.

As with laboratory measures of performance, differences between smokers and nonsmokers in physical fitness test performance are magnified with age and smoking history. Patton et al. (1982) found smoking to be one of the strongest discriminators between different levels of fitness-test performance in research on 270 over-40 military personnel (average age 43.8 yr). Although half of the total group and half of the "fair" fitness subgroup were smokers, 77 percent of the 22 soldiers with "very poor" fitness levels and 72 percent of the 59 soldiers in the "poor" fitness group were smokers. This contrasted with 29 percent smokers among the 58 soldiers scoring in the "good" fitness range, and only 22 percent smokers among the 32 soldiers in the "excellent" fitness group. A treadmill exercise tolerance test was used to classify fitness groups.

It should be mentioned that research in the 1930s, 1940s, and early 1950s was much less consistent in demonstrating reduced performance on strength and endurance tests for smokers compared to nonsmokers or as a result of smoking immediately prior to testing in habitual smokers. Few, if any, studies showed an advantage of smoking over not smoking, although four of the top five finishers of a Pittsburgh marathon were smokers (Karpovich and Hale 1951). However, studies frequently did not find a significant difference between smokers and nonsmokers or between performance while smoking and performance while not smoking in habitual smokers (e.g., Reeves and Morehouse 1950). Athletic training and participation appear to overcome many of the performance problems caused by smoking (Morton and Holmik 1985) and the smaller differences between smokers and nonsmokers in past studies may reflect the more widespread habit of cigarette smoking among athletes prior to the initial report on smoking and health of the Surgeon General (US Department of Health, Education, and Welfare 1964) and prior to much of the other evidence of the high risk to health from smoking.

Oxygen debt in smokers and nonsmokers

Oxygen debt refers to exercise effects which cause the heart rate and the respiration rate to remain at higher than normal levels following exercise. These elevated heart and respiration rates provide oxygen needed to restore muscles to the pre-exercise state. This variable of oxygen debt has been largely displaced in more recent research on human performance which obtains frequent or continuous recordings of expired gases, blood lactate levels, and other chemicals and physiological variables during exercise (Dr. James Vogel, personal communication 1984). However, earlier studies that focused on oxygen debt still provide considerable information, even if the different factors contributing to the oxygen debt are not available for separate assessment. What is more, oxygen debt will delay or impair subsequent physical efforts like assaulting the next hill, and it appears to have much tactical relevance.

Several studies (Chevalier et al. 1963, Frayser 1974, Krumholz, Chevalier, and Ross 1964) have shown oxygen debt accumulation for a given work task is appreciably higher for smokers than for nonsmokers. Smoking did not occur for at least 1 h prior to testing in these studies. This also was true in the research of Krumholz, Chevalier, and Ross (1965) who measured oxygen debt and pulmonary function before and after a 3-week period of abstinence in smokers. They found oxygen debt to be decreased significantly while lung function (expiratory peak flow and pulmonary diffusing capacity) were increased significantly as a result of the 3 weeks without smoking.

No studies of smoking and oxygen debt were found that showed an absence of differences between smokers and nonsmokers. Krumholz and Hedrick (1972) found ex-smokers and nonsmokers both showed less oxygen debt than smokers, but nonsmokers and ex-smokers did not differ between themselves. The implication is that smoking produces deleterious effects on the ability to recover from strenuous physical activity, but quitting smoking largely eliminates these deficiencies with some of the deficiency eliminated within a few d or even h. Since oxygen debt is directly related to the duration of strenuous exercise, nonsmoker advantages over smokers would be expected to increase as the duration of the period of exercise increased. Research is needed to confirm this assumption.

Magnitude of smoker-nonsmoker differences

Astrand and Rodahl (1970) estimated smoking reduces maximal aerobic power by five to ten percent. They point out

this is a serious decrement in performance of the heavy work associated with many types of athletic events, since a regular physical training program will increase maximal oxygen uptake by only 10 to 20 percent. Physical fitness test differences between smokers and nonsmokers are of similar magnitude. Cooper, Gey, and Bottenberg (1968) found young airmen who smoked more than 30 cigarettes daily, were, on the average, able to cover only 92 percent of the distance averaged by nonsmokers in a 12-min run. Kujala (1981) found a similar seven-percent decrement associated with smoking among young Finnish soldiers. He also found the number of cigarettes smoked daily was directly related to the decrement in running performance. Hartling (1975) studied performance of Danish military conscripts and found an average difference of 5.5 percent between smokers and nonsmokers on the distance run in 12 min. All of these studies represent performances of young men. The performance of older smokers differs from that of older nonsmokers by more like 20 percent (Raven et al. 1974a).

Differences in capacity for endurance training between smokers and nonsmokers

Not only are smokers at a disadvantage relative to nonsmokers in tests of physical endurance, some studies have shown cardiovascular endurance training occurs more rapidly in nonsmokers than in smokers (e.g., Cooper, Gey, and Bottenberg 1968). They found that after 6 weeks of training, nonsmokers had increased their 12-min-run distance by 11 percent, whereas smokers of more than 30 cigarettes daily had shown an increase of less than seven percent. They claimed a "... person never could achieve maximum performance or respond completely to training as long as he continued to smoke any number of cigarettes." However, Frayser (1974) found much more improvement in smokers than nonsmokers following 30 d of cardiovascular training, although oxygen debt still was slightly higher for smokers than nonsmokers following testing after training. Another exception to greater training effects for nonsmokers was the research of Pleasants (1969) who found his swimmers who smoked not only did not differ from nonsmokers on pretests, but benefited equally well from training as the nonsmokers.

As with physical performance levels, the capacity for change of performance levels may differ between smokers and nonsmokers as much for motivational or personality differences as it does for differences in physiological capacity (see Chapter 10: "Smoking, abuse of other substances, delinquency, and driving accidents"). Whether for motivational, physiological, or other reasons, Blair et al. (1984) found men who were tested at the Cooper Clinic on at least two occasions over a

period of at least 1 yr, and who were found to have improved their physical fitness, were much less apt to smoke (15.4 percent) than men who were similarly tested twice and did not show improvement between tests (28.2 percent). Two other recent studies may shed light on this association of smoking with lack of improvement. Maksud and Baron (1980) compared young smokers and nonsmokers during several levels of exercise and found perceived exertion to take higher levels for smokers than nonsmokers. A similar pattern for "minute ventilation" suggested respiratory factors may be a major factor in perceived exertion. These differences in perceived exertion occurred even though heart rate and oxygen consumption were similar between smokers and nonsmokers at all levels of exercise. Hughes et al. (1984a) also found smokers experience a higher level of perceived exertion for a given heart rate than nonsmokers. This relationship held when the increased activity of nonsmokers compared to smokers was controlled and when the reduced pulmonary function of smokers compared to nonsmokers was controlled. The implication of both studies is that exercise at a given heart rate is harder for smokers than nonsmokers. One might expect smokers to train less hard than nonsmokers since it hurts more.

Are smokers at an advantage at higher altitudes?

According to the founder (R. Fehl, personal communication 1983), the Pike's Peak Marathon had its origins as a competition between smokers and nonsmokers in 1956. Although a smoker did not win, the fact smokers were able to successfully complete this grueling race indicates smoking does not totally preclude even superb physical performance, given that proper athletic training occurs. Based on informal observations of himself and other smokers during mountain climbing, MacLean (1979) argued work at altitude may be somewhat easier for smokers since "... tissues had become so accustomed to oxygen starvation due to the cigarette smoking that we were already partially acclimatised to altitude before setting off." In the Raven et al. (1974a) research with older males, smokers showed smaller decrements in performance as a result of breathing 50 ppm CO in filtered air than nonsmokers. Their COHb levels only increased by 14 percent compared to a 200 percent increase in COHb for nonsmokers. However, although the smokers showed smaller decrements in performance than nonsmokers following CO inhalation, nonsmokers still outperformed smokers by 27 percent regardless of ambient conditions.

Fine (1968) measured the performance of soldiers on a 600-yd run that was carried out at two elevations and found decrements associated with altitude were related to the amount these soldiers smoked. These 45 Special Forces soldiers first

ran 600 yd at sea level. The next day, they were transported to a mountain research site at a 13,000-ft elevation and tested again. Smoking was not a significant predictor of these run times at altitude (probably because of a large variance between individuals). However, the correlation between amount of smoking and the change in run time from sea level to 13,000 ft was significant (r=.50), with increased smoking associated with a bigger increase in time for the run at 13,000-ft elevation. The correlation between age (which ranged from 21 to 44) and this difference in run time with altitude also was significant (r=.49). Unfortunately, no separate correlation between age and run time was reported for smokers or for nonsmokers. The results of Raven et al. (1974a) and Patton et al. (1982) showing larger effects of smoking with increased age and increased time of smoking suggest this correlation between age and performance would be greater for smokers than for nonsmokers.

Smokers have not been found to be at a disadvantage at altitude in all research. Wagner et al. (1978) compared bicycle ergometer performance of smokers and nonsmokers who pedaled bicycle ergometers in an altitude chamber at an "altitude" of 10,000 ft while breathing low levels of CO (40 ppm) that produced blood COHb levels of approximately 5 percent. Few differences appeared between smokers and nonsmokers and those that did typically indicated better adaptation of the smokers to the hypoxic stimulus. Unlike smokers who showed no heart rate and stroke volume changes at altitude plus CO exposure, nonsmokers increased their heart rate and stroke volume. Nonsmokers' subjective reactions to the work at altitude (with 4.2 percent average COHb levels) also reflected these increased physiological reactions. This was a group of young men between the ages of 22 and 34. Older subjects may not have shown this smoking advantage given the large decrements in older smokers relative to nonsmokers reported by Raven et al. (1974a).

A recent study by Lindgarde and Lilljekvist (1984) looked at the effect on 51 "installation workers" of moving from Sweden to a work site at 3,200 m in the Peruvian mountains. They found 12 of the 25 smoking workers, but only 2 of the 26 nonsmoking workers were unable to complete their 2-yr work contracts. The two nonsmokers and two of the smokers had clearly medical reasons for terminating their contracts. The basis of termination for the other ten smokers was unsatisfactory performance. Ten of the 12 smokers terminating their contracts were judged to be "over-consumers" of alcohol and the authors indicated alcohol problems may have been a factor in some of these terminations. However, they also pointed out most of the men "had previous experience of similar work abroad, i.e., installation work, at low altitudes, and they had

had no difficulty in managing the work." Unfortunately, no data on age for the successful and unsuccessful workers was provided except for a comment that many of these men had many yr of foreign experience. The contrasting results for young men in the Wagner et al. (1978) study and the older men of the Raven et al. (1974a) suggests these workers may have been middle-aged, rather than young.

Lindgarde and Lilljekvist (1984) found both smokers and nonsmokers showed large increases in hemoglobin and hematocrit when moving from sea level to permanent residence at 3,200 m. However, the rise in these blood variables was sharply higher for smokers than for nonsmokers, despite the fact that smoker's hematocrit was higher from the beginning. The average difference in hematocrit level between smokers and nonsmokers at sea level was .9. After living for a period at 3,200 m this difference increased to 3.5. These hematocrit changes also may bear on the more successful adaptation of smokers in the study of Wagner et al. (1978) and the less successful adaptation found for smokers by Lindgarde and Lilljekvist. Smokers' elevated hematocrit may be adaptive early in exposure to high elevations, but may become maladaptive when hematocrit increases to very high levels as occurred for smokers following a few weeks exposure to elevation in the study of Lindgarde and Lilljekvist. Presumably, such thick blood begins to clog the capillaries.

Muscle/strength differences between smokers and nonsmokers

This review of effects of smoking on physical performance has concentrated primarily on the differences in cardiovascular and pulmonary function between smokers and nonsmokers and on the relationship of cardiovascular-pulmonary function to amount of smoking. This is because the bulk of research is in this area. However, research by Orlander, Kiessling, and Larsson (1979) indicates some of the performance differences between smokers and nonsmokers may reflect differences in muscle structure and function that are produced by or at least are related to smoking. They found there was a lower percentage of Type I muscle fibers in the leg muscles of smokers (38 percent) compared to nonsmokers (51 percent) and a higher proportion of Type IIB fibers in smokers (26 percent) than nonsmokers (16 percent). They also found muscular oxidative capacity was significantly lower in smokers than in nonsmokers.

Orlander, Kiessling, and Larsson also measured leg strength and found it to be a significant 16 percent greater for nonsmokers than smokers. Muscular endurance, although greater for nonsmokers, was not significantly different. However, at high leg movement speeds, smokers showed advantages

in dynamic strength over nonsmokers. The average age of these men was 44, indicating long term smoking among the smokers.

Larsson and Orlander (1984) and Larsson, Gransberg, and Knutsson (1985) conducted further studies of muscle structure and muscle function between smokers and nonsmokers. To help control for body size and other physiological differences, they used identical twins who were discordant on smoking. Large differences in the proportion of Type I fibers and the proportion of Type IIB fibers again were found with nonsmokers having 52 percent Type I fibers versus 40 percent for smokers. Nonsmokers had a lower proportion of Type IIB fibers (18 percent) than smokers (29 percent). Strength differences between smoking and nonsmoking groups were less pronounced than in the Orlander, Kiessling, and Larsson (1979) study.

Ingemann-Hansen and Halkjaer-Kristensen (1978) also studied muscle structure of smokers and nonsmokers and found significant differences between the cross-sectional area of Type I muscle fibers in the quadriceps with the cross-sectional area in smokers only 86 percent of that in nonsmokers. There also was a significant negative correlation between tobacco consumption of smokers and the proportion of Type I muscle fibers. These were young soccer players who were similar in age, height, body weight, lean body weight, thigh volume, and isometric as well as dynamic quadriceps strength.

Mellstrom et al. (1982) measured grip strength of 70-yr-old men and found smokers were significantly lower on this variable than nonsmokers. Kay and Karpovich (1949) used a within-subjects design where smoking occurred during a rest period of tests of grip strength in one session and did not in another. No significant differences were found in grip strength following rest or following smoking. What is more, the differences that did appear favored the smoking condition.

Conclusions and military implications

Even young soldiers will show improved capacity for physical work with even brief abstention from smoking. Commanders wanting to maximize physical fitness test scores or physical performance in combat would do well to prohibit smoking for several h prior to testing. However, such prohibition of smoking could backfire. Some performance decrements on simulated driving tasks that will be described in Chapter 8: "The effects of tobacco deprivation", may have reflected low motivation of soldiers who were unhappy about being assigned to the no-smoking group. However, the soldier who is highly motivated to perform on the PT test will benefit from even a few h of smoking abstention.

One other relevant finding, however, is the person highly motivated for performance on the physical fitness test is much less apt to be a smoker (Biersner, Gunderson, and Rahe 1972). What is more, the higher motivation and lower perceived exertion for a given heart rate of nonsmokers during physical training will lead to greater training effects for nonsmokers and this would be expected to magnify their advantage over smokers. This could provide strong arguments for reduction of smoking in the soldier population.

However, perhaps the major implication of this chapter is that smoking speeds the process of making old soldiers. Although young smokers showed zero or small differences from young nonsmokers on laboratory tests of physical performance (unless they smoked immediately prior to or during testing), invariably as the age of the smokers and their exposure to smoking increased, their performance declined. Changes in performance with age of nonsmokers were much smaller. The studies of athletes who smoke indicate the deterioration with smoking on physical capacity can be countered effectively for considerable periods if athletic training continues at high levels.

For the most part, the research that has been conducted on smoking and physical performance has dealt with cardiovascular performance. However, some studies of muscular strength also showed poorer performance of smokers, especially older smokers.

Chapter 3

Effects of smoking on perceptual processes

Vision and other perceptual processes are critical to soldier performance. It is anticipated that future conflicts will involve continuous operations in diverse environments and weather conditions. As a result, darkness, fog, and other low-visibility conditions will severely tax the sensory and perceptual systems of the soldier, particularly, the visual system (Department of the Army 1983). Numerous studies have been conducted over the years that relate tobacco smoking and nicotine to visual performance during both laboratory and real-world tasks. These studies have included both immediate effects of smoking and differences between smokers and nonsmokers, i.e., long-term effects of smoking. These studies will be reviewed in this chapter. Contradictory results often have been reported from what appear to be similar research efforts. Even in the area of night vision where smoking has long been viewed by many researchers as deleterious, the results from different research studies are contradictory. These contradictory results indicate a large need for additional research on smoking and perception.

Smoking-related visual deficits, such as slower dark adaptation and lower final levels of visual sensitivity to dim lights that have been found in some studies (McFarland 1970), appear to be related to the carbon monoxide (CO) in cigarette smoke which increases the levels of carboxyhemoglobin (COHb) in the blood.[1] Nicotine has been blamed, however, for night-vision deficits from smoking immediately prior to testing of visual sensitivity by at least one researcher (Sheard 1946) who found smoking-produced deficits were not relieved any faster by breathing oxygen than by breathing air. Some serious visual problems, such as tobacco-amblyopia, apparently result from cyanide compounds in smoke (Dang 1981). However, these conditions are so rare they will receive little further discussion here. Differences in night vision favoring non-smokers over smokers also have been blamed on cyanide compound poisoning of the smokers (Durazzini, Zazo, and Bertoni 1975).

Enhancement of perceptual performance by smoking, when it occurs, appears to be a result of nicotine. since nicotine from other sources than smoking typically produces similar results.

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[1] See the chapter on smoking and endurance for a review of the CO-COHb relationship and of COHb effects on oxygen transport and utilization.

Increased cerebral blood flow (Wennmalm 1982) and increased macular blood flow (Robinson, Petrig, and Riva 1985) following smoking may contribute to these improvements in visual performance.

The effects of smoking and nicotine on basic visual and ocular processes first will be discussed in this chapter, to be followed by their effects on visual perceptual processes. The effects of smoking on hearing will be dealt with following the chapter on vision and visual perception.

There are two key questions that will be considered throughout this chapter as they were in the previous chapter. One question is: "What are the differences between smokers and nonsmokers?" This can be restated as, "What are the long-term effects of smoking?" The other question is: "What are the immediate (acute) effects of smoking?"

Scotopic sensitivity and dark adaptation

Effects of carbon monoxide: As was described in the chapter on the effects of smoking on physical performance, carbon monoxide (CO) is a major component of tobacco smoke that combines with hemoglobin to form carboxyhemoglobin (COHb), and this reduces the amount of hemoglobin available to transmit oxygen. Oxygen is critical for neural and sensory systems (McFarland 1970) as it is for muscle systems (Vogel and Gleser 1972), and studies of CO effects would be expected to be directly relevant to studies of the immediate effects of smoking.

COHb levels of three to ten percent, which correspond to the range of COHb levels produced by smoking (Castleden and Cole 1975), have been found to produce effects on visual sensitivity in some studies, but to show no effects in others. In a review of behavioral effects of CO on animals and man, Laties and Merigan (1979) found the bulk of studies indicating visual function was quite insensitive to the effects of breathing CO even when COHb concentrations were well above the ten percent that is nearly the maximum COHb produced by very heavy smoking. For example, Luria and McKay (1979a) found neither smokers nor nonsmokers showed changes in visual sensitivity as a result of exposure to 195 ppm CO mixed with air that raised nonsmoker COHb levels to 9 percent and smoker levels to a range of 10.2 percent to 13.3 percent.

It is of major interest that Luria and McKay did find significant and marked improvements in night vision over a 3-h testing period for smokers when breathing air. Only about a three percent drop in COHb levels occurred during this period when no smoking was allowed, and the change in COHb does not

appear to be the basis for the improvement. This is because there was no decrease in visual sensitivity with a considerably larger increase in COHb in the CO-breathing condition. Luria and McKay (1979a) do not try to account for this significant improvement of smokers in the control condition. It may be that some change in accommodation, pupil size, or other ocular variable with smoking deprivation may have influenced the improvement of nonsmokers during the 3-h testing session where air was breathed. Roberts and Adams (1969) showed accommodation and pupil size both changed with smoking and it is probable that smoking deprivation also has effects on these ocular systems. If this improvement in sensitivity over a few h of abstinence from smoking is a general visual phenomenon, it could have major implications for improving performance in darkness of soldiers who smoke.

However, a few studies have found effects of very low levels of COHb on visual sensitivity. Using highly sensitive tests, very dim targets, and trained subjects, McFarland (1970) reported significantly reduced sensitivity of the eye when COHb was increased to levels as low as three percent by breathing CO mixed with air. McFarland also reported at very high intensities, such as those produced by sunlight, oxygen lack (increased COHb) produced practically no change in foveal visual acuity.

Evidence that accommodation changes may be implicated in some of the CO effects on scotopic sensitivity comes from a study by Kobrick et al. (1984) who found extended hypoxia (relative oxygen deprivation) increased thresholds for green lights during dark adaptation compared to performance at sea level. No effect of hypoxia was found for red lights when they were presented in the adaptometer. If hypoxia or some other unique aspect of the high altitude environment changed the resting level of accommodation or otherwise changed the ability to focus at close distances, the "closer" optical distance of the shorter wavelength green lights may have led to the "hypoxia" effects.

Immediate effects of tobacco smoking: Other research by McFarland and his colleagues reported by McFarland (1970), indicated three cigarettes smoked in the space of 1 h raised foveal thresholds by about .4 log unit and at the same time raised COHb levels to 4.1 percent from .3 percent. This effect of smoking was judged to reflect the CO in cigarette smoke since COHb levels of three percent (produced by breathing reduced oxygen during the same experiment) caused a change in visual sensitivity intermediate to the visual sensitivity that resulted from smoking of the second and third cigarettes, which had produced COHb levels of 2.5 percent and 4.1 percent, respectively.

Sheard (1946) studied immediate effects on dark adaptation of tobacco smoke and smoke from cigarettes without nicotine. He reported substantial decreases in light sensitivity of both rods and cones for tobacco smoke, whether inhaled or just held in the mouth, but no decrement in sensitivity for smoke from nicotine-free cigarettes. The effect lasted for 15 to 30 min following smoking. This would appear to support the smoking-related decrement in light sensitivity found by McFarland. However, unlike McFarland, who attributed the diminished sensitivity to COHb, Sheard attributed the effect to nicotine. Although the brief report by Sheard does not go into detail, this attribution to nicotine probably was because he found breathing pure oxygen did not eliminate the reduction of sensitivity any faster than breathing air, and also because smoking nontobacco cigarettes did not produce the decrement. However, one wonders whether experimenter or subject expectations were not a major factor in Sheard's results. Not the least problem is that nicotine from cigarette smoke is poorly transmitted to the brain unless tobacco smoke is inhaled (Henningfield 1984).

On the other hand, Troemel, Davis, and Hendley (1951) found dark adaptation occurred significantly faster, and final sensitivity was greater, after smoking. A possible explanation the authors proposed for this effect was that nicotine releases glycogen, thereby facilitating the chemical processes underlying dark adaptation. Recent research suggests increased macular blood flow following smoking (Robinson, Petrig, and Riva 1985) may be another critical factor or, at least, a contributing factor. Similar results to Troemel, Davis, and Hendley were found by Gramberg-Danielsen, Puls, and Tolksdorf (1974) who reported three or four cigarettes in a 30-min period improved dark vision and increased speed of readaptation to the dark. Bohne (1962) (reported by Calissendorff 1977) also found improved dark-vision after smoking.

Calissendorff compared dark adaptation over a 20-min period in moderate smokers in conditions where they either smoked or rested and found a small, but significant decrement associated with smoking for the first 15 min of dark adaptation. However, by the 20-min point of adaptation, visual sensitivity during the smoking conditions was not different from those during rest conditions. Calissendorff also looked at the effect of smoking a cigarette after 40 min of dark adaptation with the smoking performed in darkness and the subjects blindfolded. No significant impairment (or improvement) of the final level of dark adaptation could be noticed over a 15-min period following smoking. In the earlier trials, where smoking of the cigarette produced decrements, smoking had occurred in "faint" light and apparently without any blindfold. Although Calissendorff did not suggest this explanation,

it could be that light adaptation from the lighting and smoking of the cigarette produced the reduced sensitivity associated with smoking that occurred during the first 15 min.

Johansson and Jansson (1965) measured the time to initially detect a dim light source in a study principally aimed at the effects of smoking on glare recovery. Subjects were smokers who at alternate sessions either smoked or didn't smoke during a 15-min dark adaptation period. Initial detection time of the dim light was not different between the smoking and nonsmoking conditions.

The results of McFarland (1970) and Sheard (1946), who reported a significant immediate reduction in dark adaptation proficiency as a result of smoking, conflict with the results of Bohne (1962), Gramberg-Danielsen, Puls, and Tolksdorf (1974), and Troemel, Davis, and Hendley (1951), who found improvements in sensitivity. The results of Calissendorff (1977) and Johansson and Jansson (1965) largely are nonsupportive of either a decrease or an increase in sensitivity associated with smoking. It does not appear to be possible to resolve the differences in these contrasting results pertaining to the immediate effects of smoking and nicotine on dark adaptation rates and on final levels of sensitivity. Additional research is needed to determine whether smoking and nicotine detract from, improve, or have no effect on dark adaptation rate and final absolute visual sensitivity.

Differences between smokers and nonsmokers: The sensitivity of the eye following dark adaptation was tested by Luria and McKay (1979b) in smokers and nonsmokers who ranged in age from 20 to 76. Ten smokers and ten nonsmokers were included in each of four age groups: 20-29, 30-39, 40-49, and 50-76. Subjects were dark-adapted, then required to identify the locations of dim lights presented at different peripheral locations in the visual field at a distance of 14.3 in from the eye in a device developed by Kinney, Sweeney, and Ryan (1960). Smokers showed significantly poorer performance on this scotopic sensitivity test than nonsmokers at all age groups. Age also produced a significant effect, with lower scores for the two older age groups than for subjects aged 20-39. Nonsmoking 40-49 year-olds performed at nearly the same level as smoking 20-39 year-olds. No mention was made of the length of the period, if any, prior to testing when smoking did not occur, although there was a period of dark adaptation. It is thus difficult to determine whether these smoker differences reflected effects of immediate smoking or long-term smoking.

Although research has shown dark adaptation proficiency does decrease with age (see Fisher et al. 1970 for a review),

the close 14.3-in viewing distance also may be a factor in the age differences found in this research. The ability to focus the eye for objects at different distances (ocular accommodation) declines with age and largely is gone by age 40 (Alpern 1969). This age-related loss of accommodation is known as presbyopia. It is possible the problems of the older subjects relative to the younger subjects were related to an inability to focus on the close targets due to their reduced or absent accommodative power. Presumably, vision was corrected for the stimulus distance, for each subject, but conditions of darkness would be expected to alter the accommodative state, causing it to drift outward from the close 14.3-in viewing distance toward a resting position that is typically 25 in or more (Leibowitz and Owens 1978). The resultant additional need for accommodation might have been met by young subjects, but would be less well met by older subjects due to their loss of accommodative power. This raises the possibility younger smokers were rendered "presbyopic" by smoking so their ability to focus on the close light targets also was impaired. Roberts and Adams (1969) found accommodation for close objects to be reduced immediately following smoking, and this supports this accommodation explanation of the results of Luria and McKay (1979b).

The reason for proposing this "ocular accommodation" explanation of the strong smoker-nonsmoker difference found by Luria and McKay is, with the exception of the studies described below by Young and Erickson (1980) and Durazzini, Zazo, and Bertoni (1975), such large decrements in dark adaptation associated with being a smoker have not been reported in the literature despite a long history of research on dark adaptation. In addition, some of the studies of the immediate effects of smoking, which were described above, have shown smoking to enhance both dark adaptation rate and final levels of dark adaptation. Smoking-related changes in accommodation that improved target focus could even explain these smoking benefits that have been found.

Young and Erickson found dark adaptation to be more than twice as long for smokers as for nonsmokers. This study was done by the US Army Tank-Automotive Command (TACOM) and the major purpose was to compare the effects on dark adaptation of exposure to red and blue tank interior lights. Subjects were exposed to either the red or blue lights and then were required to judge the location of a dark square against a dimly illuminated background as soon as their vision adapted to the reduced illumination and they could make this discrimination. Adaptation times averaged 12.7 s for smokers following exposure to red interior lights compared to 4.7 s for nonsmokers. Following exposure to blue interior lights, the adaptation period averaged 21.7 s for smokers and 7.0 s for nonsmokers. This large difference between smokers and nonsmokers in a simulated

tactical environment appears to indicate that smoking effects on dark adaptation are of sufficient magnitude to influence real soldier behaviors and such differences might justify selection of only nonsmokers for work in such tactical environments or temporary prohibition of smoking for soldiers who smoke if research showed such prohibition improved adaptation.

However, although the smoker-nonsmoker differences found in the study are undoubtedly real, they also may represent an effect of smoking or smoking deprivation on ocular accommodation and not a dark adaptation difference between smokers and nonsmokers. Initial adaptation took 20 to 30 min, and if smoking deprivation for that period (plus some probable earlier abstinence period) were to increase the difficulty of focusing the eyes at a distance, this could have interfered with smoker identification of the location of the dim targets since the target distance was 2.8 m. Difficulty in focusing at a distance could result if smoking deprivation caused the eyes to adopt the resting level of accommodation or "dark focus," as it is known, which averages only two-thirds of a m (Leibowitz and Owens 1978). The effects of smoking on accommodation processes are poorly understood (see below), and no studies have been conducted of the effects of smoking or the effects of smoking deprivation on the dark focus of the eye (Leibowitz, personal communication 1983).

Durazzini, Zazo, and Bertoni (1975) also found poorer night vision for smokers than nonsmokers. They found pilots who were heavy smokers took longer to adapt to darkness than nonsmokers and light smokers. These differences were correlated with levels of cyanide compounds in the blood and in the urine of these men. The investigators saw a causal relation between these cyanide compound levels and the visual deficit. Unfortunately, COHb levels were not measured by Durazzini, Zazo, and Bertoni, but these undoubtedly also would have been positively correlated with amount of smoking even as was the level of these cyanide compounds. Sufficient details were not provided on the dark adaptation task to allow any discussion about possible confounding effects of smoking or smoking deprivation on ocular accommodation.

The results of Bohne (1962), Gramberg-Danielsen, Puls, and Tolksdorf (1974), and Troemel, Davis, and Hendley (1951), who found improved dark adaptation with smoking in smokers, may not conflict with Durazzini, Zazo, and Bertoni (1975), Luria and McKay (1979b), and Young and Erickson (1980), who found large differences favoring nonsmokers over smokers. The former three studies were on the immediate effect of smoking on smokers and the other research compared smokers with nonsmokers. If subsequent research should confirm there is a long-term effect of smoking on visual sensitivity, there would be parallels in

the short-term and long-term effects of smoking on cardiovascular performance. There would be corresponding minimal effects of immediate smoking on visual sensitivity and on physical performance and corresponding larger effects of long-term smoking on both of these variables.

Even as there is a need for further research on the immediate effects of smoking to resolve contradictory results showing improved, unchanged, and decreased visual sensitivity with smoking, the smoker-nonsmoker differences in dark adaptation also need further research, especially given possible effects of smoking on accommodation which might account for the dramatic differences between smokers and nonsmokers reported by Luria and McKay (1979b) and Young and Erickson (1980). Should the results of this research indicate the substantial differences found by Luria and McKay and Young and Erickson and also indicate that they are bona fide differences in adaptation rate, there would be a strong basis for selecting nonsmokers for fighting in reduced illumination. This is certainly one of the most important research needs in the area of smoking and soldier performance.

Critical flicker frequency

The 60-Hz "flicker" of standard incandescent and fluorescent bulbs is not noticeable since this on-off rate is faster than the threshold rate for perception of flicker. This frequency at which a rapid pulsing light is first perceived as flickering is called the critical flicker frequency (CFF). The CFF varies with the brightness of the stimulus, the size of the stimulus, and the relative duration of the on and off components of the stimulus (Landis 1954). The sensitivity of the visual system also influences CFF with more sensitivity leading to a higher CFF. For example, CFF performance has been shown to be impaired by sedative drugs and improved by stimulants such as caffeine (Smith and Misiak 1976).

The effects of smoking on flicker perception have been extensively studied and most studies indicate greater sensitivity to flicker following smoking. Waller and Levander (1980) compared the performance of smokers in smoking and nonsmoking conditions. In the smoking condition, CFF testing was preceded by three puffs on a fresh cigarette in a 1-min rest period between trials. CFF threshold increased with smoking, and the relative increase in frequency at which flicker could be detected was particularly dramatic since CFF threshold actually decreased over comparable trials where there was no smoking between trials. The changes were only on the order of one cycle per s between the averages for the smoking and nonsmoking conditions on trials that showed a maximum difference.

However, these changes were highly reliable. The significant improvement following the five three-puff smoking periods apparently indicates either an increase in sensitivity of the sensory system, of the brain which processes the neural signals from the retina, or of both. Waller and Levander did not use an artificial pupil, but claimed that pupil changes were not a factor since another group of smokers showed no changes in pupil size during smoking and nonsmoking periods.[2] According to Waller and Levander, the drop in performance for the nonsmoking condition may reflect an effect of fatigue. This drop in performance will be discussed further in Chapter 4: "Effects of smoking on vigilance, rapid information processing, and divided attention."

Barlow and Baer (1967) also found significant improvement in CFF 1 min after smoking (ten puffs on a cigarette). This occurred both for light and heavy smokers. Light smokers' CFFs gradually returned to presmoking levels with a complete return in 10 min. Heavy smokers' CFFs dropped 5 min after smoking to presmoking levels. However, 10 min after smoking, CFF dropped to a level that was significantly below presmoking levels with all 15 heavy smokers showing this change. Following this, CFF rebounded 5 min later to a level significantly higher than presmoking levels, although not as high as the level 1 min after smoking. This striking biphasic change in CFF for heavy smokers is not explained readily and calls out for additional research with close monitoring of nicotine levels in the smoker, control or monitoring of pupil and lens of the eye, and longer testing periods than the 15 min following smoking used by Barlow and Baer for their study.

Warwick and Eysenck (1963) found improved CFF performance from 15 to 20 min after either smoking a cigarette or the administration of a nicotine tablet. Nonsmokers were included in this study. They were not affected by smoking, presumably because they did not inhale, but they did show improved CFF performance with nicotine tablets. Smokers did not show improvement unless they were deprived of tobacco for 12 h prior to testing. This is similar to the results of Larson, Finnegan, and Haag (1950) who also found a period of deprivation was required for the increase in CFF following smoking to appear. Unlike Warwick and Eysenck, Larson, Finnegan, and Haag found nondeprived smokers had somewhat higher initial CFFs than deprived smokers. Smoking a very low-nicotine cigarette did not change CFF, indicating nicotine was the critical determi-

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[2] This conflicts with research by Roberts and Adams (1969) and Henningfield et. al. (1983) who showed that pupil size reliably increased with smoking.

nant of the increased CFF performance following smoking found in the Larson, Finnegan, and Haag study.

The improvement in CFF with smoking occurs despite a decrease in CFF associated with CO inhalation. Seppanen, Hakkinen, and Tenkku (1977) found increases in COHb saturation above 6 percent produced a significant decrease in CFF with each percentage increase in COHb reducing CFF by .4 Hz. Smoking and nonsmoking groups were compared by them and showed no group differences at any level of COHb concentration. COHb concentrations were higher for smokers than nonsmokers during air breathing, but the difference decreased as CO breathing continued prior to each of the six testing periods. This absence of difference between smokers and nonsmokers for CFF "sensitivity" found by Seppanen, Hakkinen, and Tenkku contrasts with the earlier reported large differences between smokers and nonsmokers for scotopic sensitivity (Durazzini, Zazo, and Bertoni 1975, Luria and McKay 1979b, Young and Erickson 1980).

Although Seppanen, Hakkinen, and Tenkku found reduced CFF with levels of COHb in the range of those produced by moderate-to-heavy cigarette smoking, Laties and Merigan (1979) reported many investigations of CFF following CO exposure have not found a change in performance, even with higher blood levels of COHb. The consistent nicotine-mediated increases in CFF with smoking found in the studies reported here indicate smoking-level COHb decrements in CFF performance are considerably smaller than the nicotine boost in CFF following smoking. However, this smoking must involve a cigarette with at least a moderate level of nicotine. Leigh (1982) found smoking increased sensitivity to flicker with high-nicotine cigarettes (1.2 mg nicotine), but smoking reduced sensitivity to flicker with low-nicotine cigarettes (.1 mg nicotine). Leigh discussed this as the result of a stimulating effect of nicotine that outweighed small COHb depressant effects during smoking of high-nicotine cigarettes. However, smoking of the low-nicotine cigarette produced too small a nicotine dose to overcome the depressant effects of COHb on CFF.

Leigh administered alcohol in some trials to study its effects on CFF. Alcohol without smoking reduced CFF, but smoking combined with alcohol administration more than compensated for this decrease when long testing blocks (required for a signal detection analysis) were administered. But in the much shorter testing blocks used in later portions of the study, smoking and alcohol led to an unexplained greater reduction in sensitivity to flicker than alcohol alone. With both short and long testing procedures, smoking without alcohol increased sensitivity to flicker.

Tong et al. (1974a) studied two-flash-thresholds as a function of smoking alone, alcohol alone, and alcohol consumption combined with smoking. The two-flash-threshold (TFT) can be thought of as the CFF when there are only two flashes.[3] The TFT is measured by increasing the interval between flashes until the subject sees two flashes instead of one or decreasing this interval until the two flashes merge into one. Tong et al. found alcohol reduced sensitivity to the two flashes. Smoking increased sensitivity to the two flashes in conditions without alcohol and smoking largely overcame the depressant effects of alcohol when smoking and alcohol consumption were combined.

They also compared nonsmokers to smokers and found a higher sensitivity for nonsmokers over smokers which "... could reflect permanent differences between the groups or the depressive effect of tobacco deprivation." Their own data appear to support the former explanation because even non-deprived smokers showed lower sensitivity than nonsmokers. However, as reported earlier, Seppanen, Hakkinen, and Tenkku (1977) did not find differences between smokers and nonsmokers in their study of CFF and research on smoker-nonsmoker differences in CFF is needed to resolve this discrepancy.

Another study comparing smokers and nonsmokers on CFF was reported by Baer (1967). Baer did and did not find differences among heavy smokers, moderate smokers, and nonsmokers. His heavy smokers average CFF was almost two cycles per s greater than for nonsmokers with moderate smokers more than one cycle per s higher than nonsmokers. However, small group sizes (five) and large differences between subjects within groups, particularly for heavy smokers and nonsmokers, prevented statistically significant results. A within-subjects variable of hyperventilation did produce significant results with hyperventilation reducing sensitivity to flicker. This result was described by Baer as contrary to previous research on the effects of hyperventilation on CFF.

Visual acuity

Except for rare conditions such as tobacco-alcohol amblyopia (Dang 1981), smoking does not appear to influence central visual acuity except perhaps at very low levels of illumination as was described for some studies earlier in the section on smoking and scotopic sensitivity. For example,

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[3] However, studies of both CFF and TFT have found them to be correlated at very low levels or not at all, according to Tong et al. (1974a).

Shephard et al. (1978) examined 426 adults and found no differences between smokers and nonsmokers in corrected or uncorrected acuity as measured by Snellen test charts.

Luria and McKay (1979b) found smoker-nonsmoker differences in median refractive error were not significant in their comparison of 40 smokers with 40 nonsmokers, although a greater positive (farsightedness) correction was associated with smoking at every age group. Luria and McKay did find a significantly higher positive correction for older subjects than for younger subjects. Given this trend for smokers to have more positive spectacle correction and the significantly more positive correction for older persons, one is tempted to suggest smoking may lead to premature aging of the refractive systems of the eye. These tendencies to farsightedness with age and with smoking, support the smoking-accommodation-change explanation of the smoker-nonsmoker differences in scotopic sensitivity with close targets which was suggested earlier.

Peripheral visual acuity does appear to be sensitive to smoking. Unfortunately, some studies show smoking to increase peripheral acuity and others show the opposite effect. Scoughton and Heimstra (1975) found smokers smoking high-nicotine cigarettes outperformed both smokers of low-nicotine cigarettes and deprived smokers in a task that required identification of whether a peripheral target was moving or stationary. Nonsmokers also were included in the study and no differences appeared between deprived smokers and nonsmokers. This finding of a superior performance for the smoking group was unexpected, given earlier work from the same laboratory (Krippner 1970) that had shown abstinence from smoking to increase the size of the visual fields. Research by Johnston (1965, 1966) also showed improvements in size of the visual fields following abstention from smoking, although the very small number of subjects casts doubt on the generality of her results.

Fink (1946) investigated the effects of smoking cigarettes of high and low nicotine content on the size of normal angioscotoma (blind spots produced by blood vessels in the retina) and found the areas to increase in both smoking conditions with a larger and longer duration increase for the high-nicotine condition. However, this may not reflect diminished visual function, but increased blood flow in, and widening of, the vessels, given the results of Bettman, Fellows, and Chao (1958) who found some subjects to have increased intraocular circulation with smoking. More recently, Robinson, Petrig, and Riva (1985) studied blood flow in capillaries of the macula of the retina and they also found a significant increase in blood flow following smoking. The time course of this blood-flow increase

corresponded closely to the angioscotoma changes found by Fink, and this strongly suggests that he was demonstrating increased blood flow in the peripheral retina and not diminished visual sensitivity with smoking as he thought.

In the single study found that compared smokers and nonsmokers on peripheral acuity, Luria and McKay (1979b) did not find differences between smokers and nonsmokers.

As with the effects of smoking on night vision and dark adaptation, the effects of smoking on peripheral acuity are contradictory and more research is needed in this area. Accommodation can influence peripheral, as well as central, acuity, and possible changes in accommodation with smoking and/or with smoking deprivation may be influencing results in many of these studies. Future research should monitor accommodation levels or control them through cycloplegia or other means.

Glare susceptibility

Johansson and Jansson (1965) simulated the glare from oncoming headlights and measured the time it took for visual sensitivity to recover from the glare source. As mentioned in the section on the immediate effects of smoking on visual sensitivity, they also measured the time to initially detect a dim light source prior to presenting the glare stimulus. All subjects were smokers who either smoked or didn't smoke at alternate sessions during the 15-min period of dark adaptation that preceded each session. Neither initial time for detection of the dim light nor time required for its redetection following exposure to the "headlight" glare source were different between the smoking and nonsmoking conditions. The authors' conclusion was "... the ability to detect objects on the road, regarded from the practical point of view, is not affected by tobacco smoking." It is unfortunate the authors did not include a group of nonsmokers in their study. The results of Luria and McKay (1979b) and Young and Erickson (1980), from somewhat similar research paradigms, suggest nonsmokers might have been at a substantial advantage over smokers in this task.

Wright, Randell, and Shephard (1973) studied the effects of CO on glare recovery, as well as on other driving-related tasks, when COHb levels were in the range of levels produced by cigarette-smoking. CO-exposed subjects required a brighter stimulus following exposure to simulated oncoming headlight glare than air-breathing subjects, but the difference was not significant. Readaptation following glare exposure took an average of 50 percent longer for CO-exposed subjects than for the air-breathing subjects, but large variation existed between

subjects and this difference also was not significant using parametric tests. However, ten of 25 exposed subjects were worse on glare recovery following exposure and this was significantly more than the four of 25 air-breathing subjects who were worse on second testing. Levels of COHb in the smoking range thus do appear to hurt glare recovery, but the results of Johansson and Jansson (1965) suggest nicotine from cigarette smoke may overcome these effects in the same way that nicotine overcomes COHb effects on CFF (see above).

Smoking effects on eye movements, ocular accommodation, and the pupil

Ocular accommodation is the process by which the ciliary muscle within the eye alters the shape of the lens in order to bring objects of different distance into focus on the retina (Alpern 1969). Powell (1938) provided a summary of several studies of smoking and accommodation and reported smoking increased the speed with which accommodation changed from an object at one distance to an object at another distance. The effect was strongest for changes in focus from far targets to near targets. On the other hand, another study reported by Powell showed facilitation of accommodation for distant objects following smoking, but a slowing of accommodation for near objects. Smoking apparently occurred immediately prior to testing in both of these studies. Powell reported that when accommodation behavior was studied for 1 h following smoking, accommodation speed was found to increase immediately after smoking and then to decrease later although no specific time intervals for the increase and decrease were given. Unfortunately, insufficient description of the methodology is given to try to account for these contradictory results and Powell's explanations are unsatisfactory. Despite the problems, the Powell report is included in this review because smoking does appear to influence accommodation in these separate studies, and because such smoking effects might account for many of the contradictory results in the areas of night vision and peripheral visual acuity.

Roberts and Adams (1969) studied the effects of smoking on both ocular accommodation and the size of the pupil. Smoking one cigarette caused an average reduction of the near point (closest distance at which the person can focus the eyes) by 2 diopters in 12 male subjects who ranged in age from 19-22. Measurements of the near point were made at 45-s intervals with each measurement occurring 30 s after inhalation from the cigarette in the smoking condition. Subjects served as their own controls and, in the nonsmoking condition, accommodation amplitude actually increased by nearly one diopter on the average over ten 45-s-spaced trials where "slightly deeper

breaths" were taken at the equivalent time intervals as the smoking inhalations. The increase in the no-smoking condition is described as an effect of practice. This practice effect subtracted from the smoking effect makes the change with smoking nearly three diopters which would correspond, for example, to a change in near point from 10 cm to 14 cm or from 20 cm to 50 cm.

Koepnick, Takahashi, and Terranova (1985) did not find an effect of smoking or a difference between smokers and nonsmokers on the ability to rapidly change the focus of one eye back and forth from about one diopter to about five diopters for a period of 1 min. A card with fine print was alternately viewed at each distance with the optical distance changed as soon as the print was in focus and words were read. The number of back-and-forth cycles in 1 min was the dependent variable. At the first 1-min series of trials the smokers performed while deprived of cigarettes for 1 h. Smokers then smoked (approximately) two cigarettes during a 15-min period of continuous smoking while nonsmokers rested. Another minute of back-and-forth focusing followed. Both groups showed a significant increase in number of back-and-forth cycles from their initial 1-min periods, but smoking did not produce a different increase in number of cycles for the smoking group. However, these negative results of Koepnick, Takahashi, and Terranova may reflect the relatively low nicotine content of the Marlboro Lights and Salem Lights which were smoked by subjects in the study.

The effects of smoking on the resting level of accommodation or "dark focus" (Leibowitz and Owens 1978), have not been investigated despite greatly improved methodology (e.g., infrared optometers and laser-scintillation optometers) for objective measurement of accommodation since the work by Powell (1938) and Roberts and Adams (1969). Such research on possible effects of smoking on "dark focus" would appear to have large implications for visual performance of smokers in situations where accommodation tends to drift to the resting state. This accommodation drift occurs in darkness, fog, or while looking through windscreens in aircraft cockpits where there is very little contour upon which the eyes can focus (Leibowitz and Owens 1978). It also occurs in other situations where attention is not directed to visual stimuli, but to entoptic visual phenomena (Dyer and Allen 1968).

Although no studies were found on the effects of smoking on optokinetic nystagmus (see Chapter 12: "Needs for additional research on smoking and soldier performance"), spontaneous vertical nystagmus was reported by Neveling and Kruse (1961) (cited in Tibbling and Henriksson 1968) to occur after smoking. This effect of smoking on extraocular muscles may augur

for an effect of smoking on spontaneous activity of the (ciliary) muscle as well.[4]

Roberts and Adams (1969) found small, but significant increases in pupil diameter during smoking with the changes amounting to about .7 mm after eight inhalations (at 45-s intervals) from a single cigarette. Subjects abstained from smoking for at least 2 h prior to testing. Henningfield et al. (1983) found intravenous injections of nicotine produced sharp increases in pupil diameter immediately following injection with a peak of about a .5 mm change 30 s following injection. Pupil diameter then dropped below baseline levels. These changes in pupil diameter with smoking indicated the need for artificial pupils or other controls in research on smoking and visual processes.

Spiral aftereffect duration

The spiral aftereffect (SAE) is a movement aftereffect that follows a period of fixation on the center of a rotating Archimedes spiral. When the spiral is stopped, it is immediately seen to move in the opposite direction, presumably because of adaptation of the neurons responsive to movement in the visual field. Eysenck, Holland, and Trouton (1957) showed depressant drugs reduced duration of the spiral aftereffect and Eysenck and Easterbrook (1960) showed amphetamine, a stimulant, caused a significant prolongation of this aftereffect. Golding and Mangan (1982a) showed smoking a middle-nicotine (1.3 mg) cigarette caused the effect to continue significantly longer in comparison with a low-nicotine cigarette (0.6mg) and in comparison with a condition where no cigarette was smoked.

The McCollough effect is a color aftereffect produced by alternatively viewing a pattern of horizontal black and green (or red) lines and a pattern of vertical black and red (or green) lines. Each pattern is viewed for 5 s and total duration of viewing of these alternating stimuli is about 5 min. Subsequent to this adaptation, viewing of horizontal black lines against a white background produces a red aftereffect and viewing of vertical black lines against a white

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[4] Another likely effect of smoking-induced spontaneous nystagmic eye movements would be to alter the electroencephalogram (EEG). Although major eye-movement artifacts are typically identified and EEG recordings are disregarded for the eye-movement period, any prolonged increase in relatively high-frequency, low-amplitude vertical (or horizontal) eye movements with smoking undoubtedly would influence the frequency spectrum of scalp potentials.

background produces a green aftereffect. Unlike the SAE which lasts for several s, the McCollough effect lasts for min or h. Smoking and nicotine tablets administered after adaptation were shown by Amure (1978) to prolong the McCollough effect. However, Amure also reported unpublished results which indicated coffee (presumably, another stimulant) reduced the duration of the McCollough effect. More research is needed to both confirm these contradictory influences of different stimulants on the McCollough effect and perhaps to help explain them.

Auditory thresholds

Ibrahim and Fatt-Hi (1983) found smokers ranging in age from 20 to 50 showed significantly more hearing loss than a control group matched on age, sex, and social class. Percentages with normal hearing, conductive deafness, and perceptive deafness were 30, 21, and 49, respectively, among smokers and 83, 3, and 13, respectively, among nonsmoking controls. Perceptive deafness was diagnosed if both air and bone conduction were impaired.

Smokers also have shown significantly more hearing loss than nonsmokers in several other studies. Thomas, Williams, and Hoger (1981) found middle-aged aviators with normal hearing smoked fewer cigarettes than those with hearing loss. Chung et al. (1982) found smokers showed more noise-induced hearing loss than nonsmokers in a sample with a wide range of subject ages.

Zelman (1973) also reported smokers to have more hearing loss than nonsmokers in a study of 1,000 consecutive audiometry candidates at a VA Hospital. Differences between smokers and nonsmokers were greatest at the higher frequencies. Ages of these patients were not described. However, smoking-related hearing defects found by Weiss (1970) largely were at lower frequencies. The differences generally were small and typically the smokers were unaware of any problem with their hearing. All of the subjects in the Weiss study were over 50 and Weiss reported earlier research had indicated no smoker-nonsmoker differences in hearing for younger men. Increased respiratory problems with smokers and the eustachian tube connection of the middle ear with the respiratory track were suggested as the probable basis of these smoking-related defects.

Extra-high-frequency auditory thresholds were claimed to be particularly vulnerable to smoking by Cunningham, Vise, and Jones (1983). However, the differences found between young smokers and young nonsmokers were small and did not reach significance. Drettner et al. (1975) found little difference

between smokers and nonsmokers in hearing loss. They reported smoking appeared to be associated with hearing loss only in those subjects who had not been exposed to high levels of noise.

Tinnitus (ringing of the ears) was shown by Chung, Gannon, and Mason (1984) to be highly correlated with smoking status with smokers having more tinnitus than nonsmokers. Since smoking and hearing loss were related directly and tinnitus and hearing loss were directly related, smoking and tinnitus were examined with a control for hearing loss and the smoking tinnitus relationship was found to be explained by the correlation between smoking and hearing loss.

Marston, Sterrett, and McLennan (1980) found young smokers and nonsmokers (aged 20 to 35) did not differ in the admittance characteristics at the plane of the tympanic membrane. Smokers smoked more than 20 cigarettes daily. However, they indicated research with older subjects with a longer history of smoking still might show effects of smoking given the results of Weiss (1970), Zelman (1973), and other investigators who found more hearing loss in smokers than nonsmokers.

Dengerink, Trueblood, and Dengerink (1984) found temporary shifts in hearing thresholds produced by loud noise actually were smaller for smokers than for nonsmokers. In addition, warm environments increased the temporary threshold shift (TTS) in nonsmokers compared to cold, but for smokers there was no TTS difference as a result of temperature. Decreased peripheral blood flow as a result of smoking and as a result of cold temperatures (for nonsmokers) appear to be related to these effects on TTS.

Generally, it is accepted that persons susceptible to TTSs also are susceptible to permanent threshold shifts (PTS). For example, variables such as iris pigmentation, which are related to TTS,[5] also are related to PTS (Thomas, Williams, and Hoger 1981). As mentioned, Chung et al. (1982) found smokers showed more permanent noise-induced hearing loss than nonsmokers which appears to be contradictory to the Dengerink, Trueblood, and Dengerink finding that smoking protected against temporary threshold shifts.

Surprisingly, no studies were found of the immediate effects of smoking on auditory thresholds. Dengerink, True-

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[5] Iris pigmentation correlates with the pigmentation in the stria vascularis of the inner ear and such high pigmentation has been hypothesized to serve an angio-protective function (Thomas, Williams, and Hoger 1981).

blood, and Dengerink (1984) confounded immediate and long-term effects by having their smoking subjects smoke before testing. One might predict decrements immediately after smoking because of carbon monoxide reduction of the oxygen-carrying capacity of the blood. Lumio (1948) found the incidence of hearing loss was high (78 percent) in workers who suffered from chronic carbon monoxide poisoning. Hearing loss was found in only 27 percent of workers exposed to CO on the job, but in whom chronic carbon monoxide poisoning could not be verified. Even when hearing defects were verified, the defect was small and the patient typically was not aware of the presence of a hearing deficiency.

The studies reviewed in this section indicate hearing changes with smoking are real and indicate diminished capacity. However, these decrements appear to be similar to decrements in physical endurance with smoking in that they do not show up until exposure to smoking has occurred for several years.

Conclusions and military implications

Large contradictions exist among the results of studies described in this chapter and probably the most compelling conclusion is more research is needed to resolve controversies related to the effects of smoking on dark adaptation and final levels of dark vision, smoker-nonsmoker differences in visual sensitivity, and the effects of smoking on auditory acuity. The contradictions in the research on night vision and dark adaptation are particularly baffling. Smoking significantly improves night vision, has no effect on night vision, and significantly degrades night vision; several "comparable" studies exist which provide each of these three outcomes.

This author has grasped at a possible influence of smoking on the focussing process of the eye as a way to account for many of the confusing and contradictory results presented in this chapter. It is surprising that smoking influences on ocular accommodation have not received more attention in the past and it could be that negative results were just not published. On the other hand, the lens of the eye and its behavior are hidden and have often been neglected relative to other visual processes. Research on smoking and ocular accommodation should probably be given high priority in civilian and military research laboratories. If smoking or smoking deprivation influences accommodation, it also could have implications for pilot performance since they frequently have problems focusing at the "infinite" distance of other airborne vehicles.

The significantly worse performance of smokers than nonsmokers on dark adaptation tasks found in several studies is of a magnitude sufficient to argue for selection of nonsmokers for missions in low illumination. However, the differences here also may reflect changes in accommodation for deprived smokers and not changes in visual sensitivity, per se. More research on smoker-nonsmoker differences is needed as well as research on the immediate effects of smoking. Perhaps, studies can look at both questions by including both smokers and nonsmokers as subjects.

The marked improvement of smoker visual sensitivity following several h of abstention from smoking, which was found by Luria and McKay (1979a), is another important area needing research since the result may be an artifact of the testing situation related to accommodation changes with smoking deprivation. On the other hand, if temporary abstention from smoking really can sharply improve the night vision of smokers, commanders should exploit this fact and ban smoking for several h prior to night patrols.

Chapter 4

Effects of smoking on vigilance, rapid information processing and divided attention

As was described in the previous chapter, smoking actually has been shown to improve visual performance in some instances, although some consistent improvements, such as the ability to perceive the flicker of a flickering light source at higher frequencies, do not conspicuously relate to any military (or other) visual performance requirements. On the other hand, the consistent smoking- and nicotine-related improvements in laboratory vigilance performance described in this chapter should apply to soldier performance in situations where the soldier must maintain high levels of visual and auditory attention for long periods when there is little to be seen or heard. Of course, there is the real danger that a smoking soldier in a "traditional" battlefield environment would disrupt his own visual dark adaptation by lighting and smoking cigarettes, or worse, give away the unit's position. Nicotine gum or nicotine from some other source than burning tobacco might prove useful in maintaining soldier performance in bona fide military vigilance tasks (e.g., West et al. 1984a).

Recent research also has shown that rapid processing of information presented on CRT displays sometimes is enhanced by smoking or other forms of nicotine administration (e.g., Wesnes and Warburton 1978, Wesnes 1985). The tasks of soldiers using modern weapons systems increasingly fit this category of behavior and these research efforts will receive much attention in this chapter since they may bear directly on the operators of such high-technology weapons systems.

Vigilance tasks and other long-term tasks

Some of the most interesting research on the effects of tobacco smoking and nicotine on performance involves visual and auditory vigilance tasks where detection of relatively infrequent events over long periods is required. Research on vigilance has looked at effects of exposure to tobacco smoke and also looked at exposure to nicotine and carbon monoxide alone. As with critical flicker frequency, nicotine tends to enhance performance, CO decreases performance, but decrements from CO (at smoking levels) usually are smaller than the nicotine-based increments in performance (see below). This is indicated by the fairly frequent finding of superior vigilance performance in smoking conditions over nonsmoking conditions. Occasionally, the smoking effect has been strong enough to cause smokers to outperform nonsmokers.

In perhaps the earliest demonstration of beneficial effects of smoking on vigilance performance, Tarriere and Hartemann (1964) found smoking increased detections of peripheral signals in a 2.5-h task designed to simulate car driving. The task involved one subtask of "peripheral visual surveillance" and another subtask of "central guiding." Deprived smokers and nonsmokers showed a significant drop in the peripheral signals detected over the time period while nondeprived smokers showed little change. Results for the central guiding subtask were not reported and, presumably, did not show decrements or at least differential decrements for the different smoking groups.

Heimstra, Bancroft, and DeKock (1967) also measured sustained performance of 20 nondeprived smokers, 20 deprived smokers, and 20 nonsmokers in a simulated driving task that involved 1) keeping a model car on the curving centerline of a moving belt, 2) depressing the brake pedal as quickly as possible when a green light changed to red, 3) detecting a 1.5-s deflection of a needle on a meter, and 4) depressing a button on the steering wheel as quickly as possible when two red lights, that simulated tail lights of a car ahead, increased in brightness. The subjects were male college students and operated this driving device for an uninterrupted 6-h session. The nondeprived smokers smoked normally during this period and in fact were given two packs of cigarettes upon entering the driving simulator.

Results from the study of Heimstra, Bancroft, and DeKock (1967) indicated deprived smokers performed significantly worse on the tracking task and the meter-vigilance task than the smokers and nonsmokers. The deprived smokers also were slower on the reaction-time task and made more errors on the brake-light-vigilance task, but these differences were not significant. Nondeprived smokers and nonsmokers typically did not differ on the various tasks, although nonsmokers showed a significant decrement for the last 3 h on the meter-vigilance task that did not occur either for deprived or nondeprived smokers. Smokers were faster than deprived smokers and nonsmokers on the task of depressing the brake pedal in response to the change from green to red of the "traffic light," with this difference being maintained for each of the 6 h of the task, but these differences between groups were not significant. All groups showed some slowing of reaction time over the 6 h, with this difference from initial performance level being statistically significant for nonsmokers and deprived smokers, but not for nondeprived smokers.

Ashton et al. (1972) compared nondeprived smokers and nonsmokers on a driving simulator and found that during the period immediately after smoking there were significant

differences in reaction times to light signals between the two groups. Sometimes the differences favored the smokers and sometimes the nonsmokers, but the authors viewed all differences as probably reflecting a stimulating or alerting effect of smoking with the longer reaction times of smokers produced by anticipatory responses prior to stimulus appearance. During later stages of the simulator task when smokers became deprived smokers, differences between smokers and nonsmokers disappeared. This is somewhat different from results of Heimstra, Bancroft, and DeKock (1967) and Tarriere and Hartemann (1964) who found decrements in performance with smoking deprivation led to significant differences from nondeprived smokers late in the sessions. However, if the smokers really were performing better in earlier stages of the task than nonsmokers, as Ashton et al. suggested, then the absence of differences at later stages also would represent decrements in performance with smoking deprivation.

In research by Frankenhaeuser et al. (1971), deprived smokers showed significant increases in simple visual reaction time over an 80-min task, while smokers who smoked three cigarettes during this period showed no decrement in performance. Myrsten et al. (1972) replicated this result for simple reaction time and also found nearly identical results for choice reaction time. Simple reaction times significantly increased during the session in the nonsmoking condition, whereas they remained constant in the smoking condition. In the nonsmoking condition, choice reaction times increased over the course of the session, whereas choice reaction times significantly decreased in the smoking condition. Nonsmokers were not compared to smokers in either of these studies.

Frankenhaeuser et al. and Myrsten et al. showed nearly identical reaction times for smokers and deprived smokers early in the session, with the deprived smokers' performance gradually deviating from that of the smokers as the session went on. These results raise a question about the results for deprived smokers in the study of Heimstra, Bancroft, and DeKock. The poor performance they found for deprived smokers compared to smokers was at or near a maximum after the first 70 min, despite that deprivation did not begin prior to the 6-h simulated driving session. It appears group differences may have existed despite random assignment in their study. Another possibility is that the deprived smokers were less motivated to perform on the various driving tasks from the outset of the 6-h session, perhaps as a result of being unhappy about being assigned to this group. Still another possible explanation of the results of these studies, with their lack of double-blind or even single-blind controls, is some smokers may have been

consciously or unconsciously motivated to make performance while smoking look better than performance while not smoking.

Wesnes and Warburton (1978) reported a series of studies on vigilance (and rapid information processing) that 1) looked at the effects of smoking cigarettes with different levels of nicotine; 2) compared smokers with nonsmokers; 3) examined smoking effects on both auditory and visual vigilance tasks; and 4) compared the effects of different levels of nicotine when the nicotine was administered via tablets dissolved in the mouth. The visual vigilance task was to detect brief pauses in an otherwise continuous movement of a clock hand. In the auditory vigilance task, subjects were required to detect from a long series of bursts of static the small proportion of bursts which also included a faint tone. The durations of these auditory and visual tasks were 80 min for each. Smokers outperformed deprived smokers and nonsmokers during the later stages of these tasks (nonsmokers were included only in the visual vigilance task). Smokers smoking nicotine cigarettes also outperformed smokers smoking nonnicotine cigarettes and this showed it was nicotine, and not some other aspect of tobacco smoke, causing the improved performance. Both visual and auditory vigilance tasks showed similar effects of smoking. Smoking effects were duplicated largely by administration of tablets containing nicotine including higher performance by subjects with larger swallowed nicotine doses.

Wesnes and Warburton reported that nonsmokers did not show expected improvements in performance on a visual vigilance task when given nicotine tablets, although light smokers and heavy smokers did benefit from nicotine tablets. They discuss this as possibly being the result of adverse effects of the nicotine tablets on nonsmokers that disrupted their attention. They also make the remarkable statement that the double-blind nature of the experiment prevented them from relating such adverse reactions to treatment condition. One wonders how they related detection data to the treatment. Interestingly, Wesnes and Warburton apparently did not find any adverse effects of nicotine tablets of the same dosage on nonsmokers in their research on Stroop performance (described below).

Although the methods are described only briefly in Wesnes and Warburton, what appears to be largely a replication of that visual-vigilance-nicotine-tablet study with heavy smokers, light smokers, and nonsmokers was reported by Wesnes, Warburton, and Matz (1983). For all three groups in this later experiment, nicotine administered 20, 40, and 60 min into the experiment reduced the decrement in detection of the interruptions of movement of the clock hand over the duration of the experiment. One mg of nicotine per tablet was more effective

than the 2 mg-condition and this may have been because some subjects experienced adverse effects from a cumulative dose of 6 mg of nicotine over a 45-min period.

Mangan (1982) found smoking improved vigilance in an auditory task which required subjects to detect when tones were slightly louder (65 db) than usual (60 db). A low-nicotine (.7 mg) cigarette was smoked by one group and a middle-nicotine cigarette (1.3 mg) was smoked by the remaining subjects in the smoking condition. Each subject also performed the task without smoking with the two sessions separated by a week. Subjects smoking the low-nicotine cigarette before the task had more detections than the group smoking the middle-nicotine cigarette or the entire group when they did not smoke (for more than 2 h prior to testing). Subjects smoking the middle-nicotine cigarette had fewer false positive responses than the group smoking the low-nicotine cigarette or the entire group when not smoking. In summary, the effect of cigarette smoking, though mixed, was to improve vigilance for both nicotine conditions over the nonsmoking condition.

It is not clear why smoking had these differential effects depending on dose of nicotine in this study of Mangan. Regrettably, data from the two groups during the no-smoking condition were not summarized separately, since the groups may have had initial differences in performance on the task despite random assignment of subjects to the groups who received different doses, and this might have helped to explain the "dose-dependent" effects. Nonsmokers were not included in this study.

Waller and Levander (1980) made frequent repeated measures of Critical Flicker Frequency (CFF) over a 50-min period which led to a lower CFF on successive trials. These changes looked very much like the performance decrements found in vigilance experiments. However, three puffs on a cigarette prior to each trial not only prevented the drop in CFF, but actually led to a sharp increase in CFF, followed by maintenance of CFF performance at a high level as long as smoking preceded trials. This elimination of normal decrements in CFF performance by smoking appears to correspond to the similar preservation of performance reported in traditional vigilance tasks and the digit-sequence-identification tasks that are described later in this chapter, and is actually discussed by Waller and Levander as another instance of improved vigilance as a result of smoking. These results are much more difficult to explain by low motivation or by negative affect from nicotine deprivation, since it is not probable that this computerized forced-choice test of sensory function would be influenced by motivation or negative emotions, even as the test was not shown to be influenced by training (Waller and Levander 1980). Although pupil changes with smoking were discounted by Waller and

Levander, they still may be a factor mediating this effect as was described in an earlier reference to this study in Chapter 3: "Effects of smoking on perceptual processes."

Results of studies of the effects on vigilance of CO-administration producing COHb levels in the smoking range typically have shown no effect on performance (Davies et al. 1981), or, in a few cases, small decrements in performance compared to performance with normal COHb levels (Laties and Merigan 1979). Improved performance with tobacco smoking when it is found, thus occurs despite the higher COHb levels of nondeprived smokers over deprived smokers and over nonsmokers.

Rapid information processing tasks

Wesnes and Warburton (1978) also studied performance on another "vigilance" task which involved processing of rapidly presented visual information. This task required detection of sequences of three successive even or three successive odd digits in a stream of digits presented at a rate of one every .6 s. This task was even more sensitive to smoking than the clock test or the auditory vigilance task. Cigarettes with nicotine enhanced detection of these odd and even sequences relative to performance following smoking of cigarettes with low levels of nicotine or no nicotine. Unfortunately, nonsmokers were not compared to smokers on this task.

Taylor and Blezard (1979) found nonsmokers consistently outperformed deprived smokers on a task similar to the task of Wesnes and Warburton that required detection of sequences of three successive even or three successive odd digits in a stream of digits presented one every .7 s. Performance was measured as the change over the 50 min. Smokers smoked a cigarette just before the task. First "epoch" scores did not differ between these smokers who had just smoked and the nonsmokers. However, over the course of the 50-min task, significantly reduced performance occurred for the smokers compared to the performance of nonsmokers. Half of the subjects had their urine acidified, which speeds the secretion of nicotine from the body, and half had their urine made alkaline, which retards the secretion of nicotine (Beckett, Rowland, and Triggs 1965). Those subjects with acidic urine would thus be expected to suffer more nicotine deprivation and their performance was, indeed, significantly worse than the performance of those with alkaline urine. Unpleasant nicotine withdrawal effects experienced while performing during later stages of the 50-min task appear to be the key to these changes in performance over time for smokers relative to nonsmokers.

In another study where Wesnes and Warburton (1983) used the three consecutive odd or even digit detection task, they found smoking a high nicotine cigarette increased the hit rate relative to lower nicotine cigarettes. Reaction time for detecting these hits showed an absolute decrease for the high nicotine cigarette (1.65 mg nicotine) relative to the lower nicotine cigarette conditions (.28 mg and .7 mg). A second experiment found similar hit rate/reaction time results for two cigarettes with nicotine (.6 mg and 1.8 mg) compared to a nonnicotine smoking condition and a no smoking condition. There was no difference in performance as a result of smoking the two nicotine cigarettes despite their similar nicotine levels to the cigarettes that did produce differences in the first of the experiments. Although nonsmokers were not included in this research, the authors argue that the smoking advantages do not just reflect improvement over a "below par" deprived state of the heavy smoker subjects (all subjects smoked more than 15 cigarettes daily). They based this argument on the research where nicotine tablets prevented the decline in performance of nonsmokers that occurred without the tablets. This included the visual vigilance task described earlier (Wesnes and Warburton 1978) and another earlier experiment (Wesnes' unpublished Ph.D. thesis) using the same odd or even digit-sequence-identification task.

Wesnes and Warburton (1984) again used the three consecutive odd or even digit detection task and found smoking of two high nicotine cigarettes (1.7 and 1.5 mg nicotine) increased the hit rate relative to lower nicotine cigarettes (.9 mg and 1.3 mg) and also relative to a no-smoking condition. Gains in hit rate occurred for smoking of all four cigarettes during the first 10 min after smoking. This was followed by a slight drop in hit rate for subjects smoking the higher nicotine cigarettes and a larger drop in hits following smoking of the lower nicotine cigarettes and in the no smoking condition. Reaction time was significantly faster than for the presmoking baseline condition for the three higher nicotine cigarettes, but showed a slowing from baseline with the .9 mg cigarette condition and the no smoking condition. As with hit rate, the largest improvement in reaction times occurred for the first 10 min following smoking with a slight slowing of reaction time performance for the second 10 min following smoking.

Edwards and Wesnes (1982) replicated the Wesnes and Warburton (1978) results favoring smokers over deprived smokers on the task involving detection of odd or even digit triads with another 18 subjects who also were habitual smokers. Cortical evoked potentials also were measured and the latency of the "P300" component of this averaged waveform was found to be shorter for smokers than for deprived smokers. Edwards and Wesnes claim their evoked potential result "implies that the

effects of smoking are not simply confined to improved regulation of sensory input, and provides support for the common self-report by smokers that smoking helps them concentrate." These results were in a brief research abstract which gave few methodological details.

Edwards et al. (1985) do not refer to the Edwards and Wesnes (1982) abstract, but it is probable that this is an expanded report of the study described in the 1982 abstract. Nineteen subjects provided data for reaction time, hit probability, and error data. However, only 12 of these subjects provided complete data on the cortical evoked potentials. Significant speeding of the "P300" component of the evoked potential occurred, but only for the first 10 min following smoking and only for the high nicotine cigarette (1.5 mg nicotine). Results for hit probability and reaction time were very similar to those reported in earlier studies with improved hit probabilities for the .9-mg and 1.5-mg nicotine cigarettes compared to the no smoking condition and with faster reaction times for the 1.5-mg nicotine cigarette condition compared to performance prior to smoking.

Edwards et al. found only a small and nonsignificant correlation ($r=.23$) between latency of the manual response (reaction time) and latency of the P300 component of the averaged evoked potential. This small correlation is claimed to be a predicted result due to the fact that their instructions emphasized both speed and accuracy during responding to the digit triads. However, such a prediction of a small correlation is like a prediction of only a small amount of pregnancy and contradicts their more critical predictions that both response latencies would be speeded by the nicotine treatments in direct proportion to nicotine dose. These two predictions logically imply a positive correlation between the two response measures and the failure to find a significant correlation probably is because of the minimal effect of smoking on the P300 component of the evoked potential.

Wesnes (1985) provided a preliminary report on a large series of further experiments that looked at the effect of nicotine on performance in vigilance and rapid information processing tasks. This series of studies showed smoking not only sharply reduced decrements in performance over time that appeared without smoking, but in some experiments smoking actually improved performance above presmoking levels.[1]

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[1] However, this could be considered just an improvement of smokers whose performance is degraded due to smoking deprivation since the typical procedure for Wesnes experiments is to have subjects refrain from smoking overnight.

In some of these three-odd-digit- or three-even-digit-identification tasks, smoking was allowed during the task (with brief breaks provided in the tasks for lighting and puffing of cigarettes). During the smoking period, performance was significantly higher than during conditions without smoking. Both detection probability and reaction time showed parallel improvements. When performance was analyzed min by min, the smoking improvement shows up in the very first min of smoking. However, this fine temporal analysis of the data showed performance during this period of smoking was restored only to the performance level that occurred during the first min of performance on the task. Smoking did not actually improve performance above these initial levels.

Most of Wesnes' (1985) previously unreported studies using the digit-sequence-identification task used heavy smokers who smoked cigarettes with at least moderate levels of nicotine. When smokers were used as subjects who normally smoked low-nicotine cigarettes, it was found that decrements in performance over time on the task in nonsmoking conditions were much smaller than for smokers who regularly smoked cigarettes with higher levels of nicotine. Wesnes (1985) interprets this as reflecting differences between the two types of smokers with the low-nicotine group not needing as much nicotine to maintain performance. However, another explanation is that low-nicotine cigarette smokers experience fewer interfering withdrawal symptoms following time on the task without smoking. It is unfortunate that groups of nonsmokers were not included more often in these studies to help clarify whether or not decrements were a result of withdrawal from smoking and whether or not improvements observed with smoking were primarily related to the elimination of such smoking-withdrawal effects.

Tong et al. (1977) compared three separate groups of nondeprived smokers, deprived smokers, and nonsmokers on an auditory "vigilance" task where subjects were to respond when they heard three odd digits in a row. One digit was presented per s for 12 min. This 12-min series was repeated five times with a 2-min rest between 12-min blocks. Tong et al. found nonsmokers outperformed deprived smokers who, in turn, tended to outperform smokers who smoked prior to the task. The nondeprived smokers did improve over blocks, but still performed significantly worse than nonsmokers on every block. These results for smokers are contrary to many of the studies showing higher performance for nondeprived smokers. Tong et al. account for the improvement of the nondeprived smokers by suggesting that, due to state-specific learning effects, smoking during the training period (of the nondeprived smoker group) may have caused the poorer performance in Block 1 for smokers when they could not smoke. However, performance was no better in Block 2 than in Block 1 for this group and the 12-min

Block 1 would have provided even more training on the task under conditions without smoking than nonsmoking smokers received in their 5-min practice trial under no-smoking conditions.

The presence of rests between blocks of stimuli in the Tong et al. (1977) study may be the key to their failure to find smoking to enhance smoker performance over the performance of deprived smokers and nonsmokers. They reported that the 2-min rest between 12-min blocks " ... was inserted since several subjects in a pilot study found the continuous task too demanding and terminated testing." Smoking reduces aggressiveness (Cherek 1981) and generally appears to be a calmer of the emotions (Gilbert 1979). Frankenhaeuser et al. (1971) and Myrsten et al. (1972) found subjective ratings of "bored," "irritated," and "concentrated," indicated the smoking condition "... tended to counteract the disagreeable feelings induced by the experiment." Smoking particularly may facilitate performance when a task is unpleasant due to difficulty, long duration, or other reasons. Making conditions easier by providing rest periods, as was done by Tong et al., may have reduced the smoker advantage that frequently has been found in vigilance and rapid information processing studies (Wesnes and Warburton 1978).

Stroop Test performance and performance on other divided attention tasks

In the Stroop Color Word Test (Stroop 1935), words are displayed in color and the task is to name this color as quickly as possible. These color-naming responses show substantial delays (and namers often show substantial emotion) when the word is a color name incongruent to the color in which it appears (e.g., the word red in blue ink--the correct response is "blue"). Stroop interference is not easily overcome by practice or other means (Dyer 1973). However, Wesnes and Warburton (1978) found nicotine administered by tablet reduced interference on the Stroop Color Word Test in both smokers and nonsmokers. This result is noteworthy and would seem to indicate a strong facilitation of selective attention by nicotine.

However, nicotine tablets did not reduce Stroop interference in a later study of nonsmokers by Wesnes and Revell (1984) in which the Stroop test was administered only a single time to subjects. Wesnes and Revell (1984) reexamined the earlier Stroop and smoking data of Wesnes and Warburton (1978) and found it was only on the second of two successive administrations of the Stroop test that the nicotine reduction of Stroop interference occurred. They concluded the effect of

nicotine tablets on nonsmokers is not a boost of Stroop performance above normal levels, but the effect of nicotine is to overcome fatigue-based decrements resulting from extended testing. The nicotine facilitation of Stroop found by Wesnes and Warburton thus would be another example of nicotine preserving "vigilance" performance.

Myrsten, Elgerot, and Edgren (1977) found deprived smokers did not show as much improvement on a second administration of the Stroop Test as nondeprived smokers. The test initially was administered to both groups during a period of smoking. The second administration was during a period of abstinence for the deprived group and smoking for the smoking group. Although the Myrsten, Elgerot, and Edgren finding did not quite achieve statistical significance, this advantage for smokers over deprived smokers tends to corroborate the finding of Wesnes and Warburton (1978) of improved Stroop performance following administration of nicotine tablets. In light of the Wesnes and Revell (1984) discussion of nicotine as a reducer of Stroop-test "fatigue," it is probably no coincidence that it was also the second Stroop Test administration by Myrsten, Elgerot, and Edgren that showed the nearly significant difference between nondeprived smokers and deprived smokers.

Suter (1981) did not find smoking to improve performance on a complex modified Stroop task in which three "colors" appeared on each slide (word denoting a color, "ink" color, and background color) and where the task was to name the complementary color of each of these three. This protracted task is much different from the more typical Stroop procedure used by Wesnes and Warburton (1978). No contradiction appears to exist between the two results.

The effects of distracting stimuli on performance also were described by Knott (1978a, 1978b). Medium intensity noise caused a small, but significant increase in simple reaction time to a visual stimulus for deprived smokers, whereas nondeprived smokers showed no difference between the noise and no-noise conditions. In a no-noise condition, the smokers and deprived smokers did not differ. Unfortunately, nonsmokers were not included in this research to determine whether or not the noise would reduce their performance as well as the performance of deprived smokers who may have been particularly prone to distraction because of unpleasant withdrawal symptoms. However, like the Stroop results, these results suggest smoking assists people in selectively attending to stimuli. Again, the effect may be more related to preservation of selective attention in tasks of longer duration than to an absolute improvement of selective attention (Wesnes and Revell 1984).

However, Friedman and Meares (1980) found lower auditory evoked potentials following smoking and higher visual evoked potentials following smoking. The diminished sensitivity this suggests following smoking for auditory stimuli also could account for the effects of noise on simple reaction time reported by Knott (1978a, 1978b). Decreased peripheral blood flow from smoking (Dengerink, Trueblood, and Dengerink 1984) may contribute to the auditory evoked potential changes.

Incidental learning is another way of examining division of attention. Andersson and Hockey (1977) found incidental learning was less for smokers who smoked a single cigarette before an immediate memory task than for another group of smokers in a nonsmoking condition. The primary task was to remember eight words presented in sequence. The incidental learning was the position on one of four corners of the screen where the word to be memorized was presented. They claim this indicated greater attentional selectivity during smoking which was related to the increased arousal produced by the nicotine. A similar explanation may apply to the reduced Stroop interference found by Wesnes and Warburton (1978) in the last half of their Stroop task.

Peeke and Peeke (1984) looked at the effects of smoking on word learning when subjects were not informed they would be asked to recall words. The announced task was word classification and the incidental learning task was measured by later recall of the words. Smoking had only a marginally significant effect on the incidental task, but contrary to Andersson and Hockey, this marginal effect of smoking was to <u>increase</u> incidental learning. The effect appeared only for those words that produced "no" responses on the classification task. Both high- and low-nicotine cigarettes caused higher learning by subjects of "no" response words compared to the no-smoking condition.

Leigh, Tong, and Campbell (1977) studied the effects of smoking (and drinking) on the ability to divide attention to different auditory information presented to different ears. They found smoking of two 1.3-mg nicotine cigarettes in a 15-min period before the task significantly facilitated the ability to count the number of clicks presented to the right ear during a 3-s burst of noise presented to the left ear. This facilitation was relative to conditions where no smoking occurred. Smoking of three cigarettes facilitated a more difficult divided attention task where right ear clicks again were counted and where the location of a 1-s 1000 Hz tone in the 3-s noise period also was identified. The tone also was presented to the left ear and was presented in either the first, second, or third s of the 3-s white noise burst. When alcohol was ingested in some conditions of this experiment, its

effect on performance was generally opposite to that of smoking.

Wesnes (1985) reported a recent experiment involving two digit-sequence-identification tasks which occurred simultaneously with one involving visual presentation of digits and another auditory presentation of digits. One hand was used to report auditory "targets" and the other to report visual "targets." Smoking had the effect of improving performance on both tasks.

In summary, smoking appears generally to improve performance in these divided-attention tasks, if, and when, they are of substantial duration. However, improvement in selective attention does not seem to be the specific mechanism since division of attention also is facilitated, particularly if instructions specifically include doing two things at once.

Conclusions and military implications

Smoking does appear to improve performance of habitual smokers on a variety of protracted tasks including simple reaction time, detection of infrequent visual and auditory events, detection of complex sequences of digits, reaction times for such detections, and tasks involving divided attention and response conflict. Nicotine administered by tablet even has been shown to prevent declines in performance of nonsmokers on some of these tasks. However, there are results from some laboratories that do not show this better performance of nondeprived smokers over deprived smokers or (in a few cases) nonsmokers. What is more, the failure of Wesnes (1985) to see differences in smoking withdrawal symptoms as the obvious explanation of differences in performance between deprived heavy smokers and deprived light smokers makes one worry about experimenter biases in some of the research showing nicotine and smoking benefits.

Military tasks such as manning a listening post frequently involve waiting for infrequent visual and auditory events. The soldier who smokes on watch in combat probably will be quickly "overcome by events." However, given the results of research on the effects of smoking and nicotine on laboratory visual vigilance tasks, the soldier who used a nonburning source of nicotine very likely would be more apt to detect the enemy or to detect the enemy more quickly than his nicotine-deprived colleague. This would be particularly true if he were a smoker, as many soldiers are. However, before administering nicotine to sentries, research is needed to study the effects (and possible negative sideeffects) of use of nicotine gum or nicotine aerosols on watch-keeping performance of actual

soldiers in simulated combat settings. Both smokers and nonsmokers should be included in this research. Control groups using placebo gum or placebo sprays are needed in double-blind research paradigms to prevent any possibility of conscious or unconscious biases related to tobacco use of subjects and experimenters from influencing results.

The rapid information processing tasks of operators of Army systems like Patriot and Aquila are not all that different from the laboratory tasks in which performance typically is facilitated by smoking, especially for nondeprived heavy smokers compared to their deprived counterparts. Research on task performance of actual Army weapon system operators such as Patriot operators needs to be conducted where nonsmokers are compared to smokers and where deprived smokers are compared to smokers not deprived of nicotine. Again double-blind paradigms using nicotine tablets or aerosols are preferable to the more typical experiments where smokers have little doubt they are receiving or not receiving a dose of nicotine.

Chapter 5

Effects of smoking on cognitive processes

Learning, memory, problem solving, and time estimation are all critical for effective performance by military as well as civilian personnel. The same questions raised relative to physical performance and to perception arise for these cognitive processes. Do smokers differ in their capacity for these activities from nonsmokers? What are the immediate effects of smoking on these activities for smokers? What is the effect on these processes when habitual smokers are deprived of tobacco? Unfortunately, only a small amount of research is available on the effect of smoking on these cognitive processes and often the research that exists does not deal with the specific types of learning, memory, and problem-solving tasks faced by the soldier and his leaders. However, this chapter will review current research and, where possible, relate these results to military tasks.

Learning and memory

Carbon monoxide effects: Although much literature exists on human performance and animal performance under conditions where CO-air mixtures were inhaled and carboxyhemoglobin (COHb) reduced the oxygen-carrying capacity of the blood, no studies were found that looked at the effects of CO on human learning and none were reported in a recent review (Laties and Merigan 1979). Unlike for the areas of sensory perception and physical performance, there is thus no baseline data for the effects on learning of this key active component of cigarette smoke. Presumably, exposure to high levels of CO would interfere with learning once high levels of COHb were attained. However, increased cerebral blood flow immediately following smoking (e.g., Kuhn 1967) could reduce or even reverse detrimental effects of the low COHb concentrations produced by smoking.

Nicotine effects on learning: There is considerable literature on the effect on learning of this highly active component of cigarette smoke and nicotine in small doses has frequently been found to have a facilitating effect on learning in rats (e.g., Bättig 1970, Bovet-Nitti 1966). Injections of nicotine after the learning trials also have been shown to improve maze-learning and this suggests nicotine facilitates the consolidation of memory as well as improving initial formation of memory traces (Garg 1969). However, Fleming and Broadhurst (1975) found no effect of nicotine on two-way avoidance conditioning in rats and also discussed several other instances where nicotine failed to promote learning or memory consolidation.

However, even if nicotine always improved animal learning, animal research provides incomplete and often unsatisfactory information about the effects of nicotine on human learning. One reason is the different forms and dosages in which nicotine is administered to animals compared to the way it is obtained from smoking. Probably of more importance, however, are the large differences that exist between human and animal learning processes. Even simple human learning involves strategies and tactics that are not available to laboratory animals.

Immediate effects of smoking on learning and memory: Memory-span refers to the largest number of rapidly presented digits that can be repeated correctly immediately after presentation. In an early study of human learning and memory as a function of smoking, Hull (1924) showed smoking immediately reduced memory-span for a series of digits for both smokers and nonsmokers. Although statistically significant, the difference amounted to only a fraction of a digit for each group. Hull also found smoking to have an immediate detrimental effect on learning of associations between geometric figures and nonsense syllables. This difference disappeared on subsequent tests later in the hour following smoking.

In the extensive series of experiments by Hull, subjects were blindfolded and either smoked a lighted pipe of tobacco, or, in the control condition, inhaled electrically heated air through the pipe. Following this, the blindfold was removed and testing began. Both smokers and nonsmokers were exposed to both "smoking" conditions and Hull claimed the subjects did not know when they were smoking a pipe with real tobacco or simply inhaling heated air. Presumably, this eliminated effects associated with expectations subjects may have had related to facilitating or detrimental effects of smoking. It reflected a much more sophisticated single-blind approach to smoking research than preceding efforts and also many later efforts. However, it seems to this ex-smoker that subjects with smoking experience would not be fooled by heated air sucked through the pipe unless they did not inhale the heated air or the smoke (which would greatly reduce any ingestion of nicotine or carbon monoxide into the body or brain in the smoke condition). Subjects without smoking experience would be even more apt to notice when smoke or heated air entered the lungs given the typical strong reaction to inhaled tobacco smoke by nonsmokers. Hull's results may be valid indications of the effects of smoking and not smoking, but they probably are not devoid of effects of subject's expectations about tobacco effects, despite "testimonials" by some subjects to the lack of difference between the smoking and control conditions. Hull's comparisons of smokers and nonsmokers are not subject to this criticism, only his claims for the immediate effects of smoking on each of these groups. His heated-air condition should

probably be considered as a sham-smoking condition such as when subjects puff on an unlighted cigarette (Williams 1980) as a control for smoking conditions.

Williams, like Hull, looked at the effect of smoking on immediate memory for digit strings. Performance was tested as a function of the nicotine content of cigarettes smoked prior to the memory task. Cigarettes with .6 mg, 1.3 mg, and 1.8 mg of nicotine were smoked on different d using a repeated-measures design. On one d an unlighted cigarette was puffed prior to the memory task. Six strings of nine digits were presented auditorily with one digit presented each s. A signal indicated when all nine digits were presented and subjects were given 11 s to record them in their correct order. Like Hull, who found an immediate decrement in memory-span following smoking, Williams found the number of errors was directly related to the nicotine content of cigarettes and the least errors occurred for the condition where subjects puffed on an unlit cigarette. These same subjects showed significantly improved performance on a letter-cancellation task as a function of smoking. This letter-cancellation task occurred during the same testing session as the immediate-memory task. The different effects of smoking on the two tasks were explained by Williams in terms of different effects of arousal on simple and difficult tasks with arousal judged higher for higher nicotine intake (unlike Kleinman, Vaughn, and Christ [1973] who explained better performance during simple tasks of deprived smokers on the basis of their heightened arousal relative to nondeprived smokers--see below).

Andersson and Post (1974) showed serial learning of a list of 30 words was significantly slower immediately following smoking of a nicotine cigarette compared to learning following smoking of a nicotine-free cigarette. Subjects were light smokers (less than six cigarettes daily) and served as their own controls in the 30-trial learning task. Subjects smoked two nicotine cigarettes while learning one list and two nicotine-free cigarettes while learning another. Subjects received the initial ten learning trials in a smoking-deprived condition (each trial lasted 2 min), then were given a break in which they smoked one nicotine cigarette (2.1 mg) or one nicotine-free cigarette. Ten more learning trials occurred after smoking the first cigarette. This was followed by another break and a second cigarette of the same nicotine content was smoked. Correct anticipations of each next word in the 30-word series were recorded on each trial.

Andersson and Post found learning for the two conditions was nearly identical until the first cigarette was smoked. On the next trial, performance dropped slightly for the nicotine condition, then resumed its upward trend on subsequent trials.

No such drop in performance occurred following smoking of the nonnicotine cigarette and performance following smoking of this cigarette was significantly better for the ten trials that occurred between cigarettes. However, performance following smoking of the nonnicotine cigarette began to level off even before the second cigarette was smoked, whereas performance following smoking in the nicotine condition continued to rise throughout the ten trials between cigarettes. Following smoking of the second cigarette, performance improved for the nicotine cigarette and within two trials the difference in number of learned words between regular and nicotine-free cigarettes had practically disappeared. In other words, learning performance of the high-nicotine smoking condition, which was inferior to the low-nicotine smoking condition for each trial following the first cigarette, actually caught up to the low-nicotine condition on trials shortly after smoking the second cigarette. If smoking one cigarette is bad for learning, apparently smoking a second cigarette is good.

Andersson and Post (1974) found heart rate elevation occurred following smoking of both nicotine cigarettes, but this elevation was considerably less for the second nicotine cigarette than for the first. The authors explain the different effect on the subjects of the two cigarettes on the basis of this fact that the first cigarette produced high arousal combined with a theory that high arousal strengthens the memory trace, but temporarily inhibits immediate recall (Walker 1958). Although this may account for the drop in performance on the first trial following the first nicotine cigarette it does not mesh with enhanced learning following the second nicotine cigarette (which also elevated heart rate). Perhaps a better explanation is that one effect of nicotine is to reduce learning ability, but another effect of nicotine is to preserve motivation for the arduous task of learning a 30-syllable list. In this explanation, a fatigue-reduction effect of smoking eventually comes to outweigh the smoking-based interference with learning in the later stages of the learning session.

In a later study by Andersson (1975), ten male subjects who were moderate smokers (5-15 cigarettes daily) participated in both smoking and no-smoking conditions during learning of a list of 25 nonsense syllables. She again found smoking during an 8-min break after the tenth learning trial significantly reduced correct anticipations on learning-test trials immediately following smoking compared to performance on the same trials when they followed a comparable 8-min break without smoking. The difference between no-smoking and smoking conditions (which favored not smoking) was diminished on trials 16 to 20. A 45-min pause occurred prior to Trial 21 which was the last trial. On Trial 21, recall was found to be slightly

higher for the smoking condition than for the no-smoking condition although the difference did not approach statistical significance except when performance on this last trial was expressed as a percentage of performance on the single trial that preceded the 8-min break (Trial 10). Even with this inappropriate transformation of Trial 21 data, the significance of the t-test comparing smoking and no-smoking conditions was greater than .05 and less than .10. Given the appropriate counterbalancing of the order of smoking and no smoking conditions across the ten subjects, there is no a priori reason why such an adjustment of the data on the basis of Trial 10 scores should occur and Andersson's claims that recall was higher after a 45-min delay for the smoking condition are unfounded.

Unfortunately, this "finding" of improved delayed recall following smoking by Andersson (1975) has been promulgated in numerous research reports and reviews (e.g., Peeke and Peeke 1984, Pomerleau and Pomerleau 1984) despite the lack of any evidence for it. These results of Andersson, like those of Andersson and Post (1974) were "interpreted" in terms of the theory advanced by Walker (1958), that high arousal facilitates memory consolidation, but inhibits immediate recall. The results of Andersson and Andersson and Post do support an inhibiting effect of smoking on learning or immediate recall, but do not support facilitation of memory consolidation.

However, Andersson and Hockey (1977) found no difference on a serial learning task as a function of smoking or not smoking prior to the single presentation of the eight-word list. This was true for words recalled in order and words recalled in any order. It is not clear why smoking did not detract from immediate memory performance on this task as it did in the earlier studies of Andersson and Post (1974) and Andersson (1975). However, the single presentation of a shorter list of words is a key difference from the repeated presentations of longer lists in earlier studies and may somehow account for an absence of a deleterious smoking effect.

Andersson and Hockey (1977) did find the incidental learning of the position of the words (which could appear in any of the four quadrants of the projection screen) was significantly higher in the condition where smoking did not occur prior to the learning task. Smoking is seen by Andersson and Hockey as reducing the subject's attention to irrelevant information even as other conditions that increase arousal (e.g., noise) lead to more focussed attention on relevant cues (see Chapter 4: "Effects of smoking on vigilance, rapid information processing, and divided attention").

Andersson and Hockey (1977) required list and position learning in a later portion of the experimental session. With both tasks specified beforehand, smokers were not inferior to deprived smokers on the word position task, and both groups did much better on position learning than in the first list where instructions did not pertain to position learning. Differences between smoking and nonsmoking groups did not differ significantly on ordered recall, recall in any order or word position for this second eight-word list again indicating an absence of deleterious effects of immediate smoking on immediate recall.

A study of the effects of smoking on learning by Houston, Schneider, and Jarvik (1978) used different groups who smoked either nicotine or nonnicotine cigarettes with the task being the learning of a 75-item list of words which were read at a rate of one every 2 s. Following the list, the subject was given 3 min to recall as many of the words as he could in any order. One "presmoking" study-test trial (list-reading followed by recall) was followed by the smoking of the cigarette (either nicotine or nonnicotine) which was followed by three additional study-test trials.

Houston, Schneider, and Jarvik found the two groups showed no significant difference in performance on the presmoking trial, with the group that would smoke the nicotine cigarette actually performing somewhat higher on recall than the group that would smoke the nonnicotine cigarette. Immediately following smoking, significantly more words were recalled by the nonnicotine group and this result corroborated the similar finding of the Andersson and Post (1974) and Andersson (1975) studies. Recall was measured again 2 d later. To control for possible state-specific-learning effects, half of the original nicotine group smoked a nonnicotine cigarette prior to recall and the other half again smoked a nicotine cigarette. Similarly, half of the nonnicotine group smoked a nicotine cigarette and half smoked a nonnicotine cigarette. State-specific effects did not appear and the recall data were directly related to the results for cigarette nicotine content from the original session. The group who originally smoked nonnicotine cigarettes had higher memory performance than the group who originally smoked nicotine cigarettes. The magnitude of the difference between nicotine and nonnicotine groups also was highly similar to the difference between these groups found on the last trial of the original session. This failure to find enhanced delayed recall following nicotine cigarette smoking over the nonnicotine condition is contrary to the reported result of Andersson, but actually supports the findings of her study.

Hrbek et al. (1973) in a briefly described study apparently found improvement of association learning 15 min follow-

ing smoking, but after 75 min subjects in the placebo (denicotinized cigarette) condition outperformed subjects in the nicotine cigarette condition. These results also are opposite to the Andersson (1975) initial decrement in learning following smoking and her claimed improved delayed recall.

The study by Houston, Schneider, and Jarvik (1978) included controls for state-specific-learning effects, but apparently did not find such effects when they looked at delayed recall as a function of smoking cigarettes with and without nicotine prior to list-learning.[1] This contrasts with the findings of Peters and McGee (1982) who specifically looked for and found state-specific-learning effects. They studied learning of a list of words as a function of smoking of a "high"-nicotine (1.4 mg) or very-low-nicotine (.2 mg) cigarette immediately prior to the learning task. Subjects viewed the 15-word list one time. The words were projected individually at the rate of one every 2 s. They then were asked to write down as many of the words as possible in any order. This immediate recall did not differ as a function of cigarette nicotine content unlike the results of Houston, Schneider, and Jarvik who found a significant advantage for a nonnicotine smoking condition.

On Day 2 of the task, subjects in the study of Peters and McGee either smoked the same kind of cigarette or the other cigarette prior to another attempt to recall the 15 words presented on Day 1. The proportion of initial words recalled on Day 2 was higher if subjects smoked the same type cigarette on Day 1 as on Day 2. This difference was significant when H-H (.86) and L-L (.88) were compared to H-L (.58), but not significant when H-H and L-L were compared with L-H (.75). Recall for the H-L combination was significantly worse than for L-H. Following the recall measure, a recognition memory task occurred where the initial 15 words that had been presented were included with 15 new words. State-specific effects did not appear for this recognition memory test although recognition for H-L was significantly poorer than for L-H.

The finding by Peters and McGee of poorer recall and recognition memory when learning occurred following exposure to high levels of nicotine, but memory was measured after exposure to very small amounts of nicotine, suggests that learning things while smoking will require smoking during recall to maximally retrieve the material. Pity the poor student who smokes while he studies, but cannot smoke during the test.

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[1] State-specific results were not central to their hypotheses and were not presented in detail.

Gonzales and Harris (1980) compared groups of deprived and nondeprived smokers on immediate and delayed recall in a learning task similar to that used by Houston, Schneider, and Jarvik (1978), although words were presented visually instead of orally. Gonzales and Harris appear to have found an advantage for deprived smokers in both initial learning and delayed recall similar to the advantage for deprived smokers in Houston, Schneider, and Jarvik. However, the small group sizes (five deprived and five nondeprived smokers) and the large and nearly significant (despite small numbers) initial learning differences favoring the deprived smokers over the nondeprived smokers prior to the deprivation/smoking treatment, complicate interpretation of these results.

Mangan (1983) compared the paired-associate-learning performance of smokers in different sessions where they either did not smoke, smoked a .7 mg-nicotine cigarette, or smoked a 1.3 mg-nicotine cigarette. Mangan found paired-associate learning was impeded by smoking nicotine cigarettes compared to a no-smoking condition when there was little interference among words in the lists. However, with high interference word lists (e.g., white paired with miss and black paired with take) smoking facilitated paired-associate learning. The decrement in performance for the low-interference list and the improved performance for the high-interference list was largely the result of the 1.3 mg-nicotine cigarette. With both low- and high-interference lists, recall by subjects 30 min after learning the list was best for the smoking conditions with the high-nicotine-cigarette condition showing the fewest errors. Smoking apparently did not occur during this 30-min wait and the subjects would have been deprived smokers for this recall session.

Mangan also compared serial learning for conditions involving no smoking and smoking of .7 mg- and 1.3 mg-cigarettes. Learning of words at the middle and end of the 20-word list did not differ as a function of nicotine conditions, but for learning of the first four words of the list, both smoking conditions were superior to the no-smoking condition. This "primacy effect" led Mangan to conclude "... the facilitatory effect of smoking is revealed in long-term, rather than short-term memory functioning."

Deprived smokers outperformed nondeprived smokers on an easy paired-associate learning task in research by Kleinman, Vaughn, and Christ (1973). However, parallel to the results of Mangan, when the task was more difficult (low association value of words instead of high association value) nondeprived smokers outperformed deprived smokers taking about 25 percent fewer trials than deprived smokers to learn the list. Kleinman, Vaughn, and Christ also compared nonsmokers in this independent

groups design and found their performance to be highly similar to the nondeprived smokers. Kleinman, Vaughn, and Christ account for the poor performance of deprived smokers on difficult tasks and their high performance on easy tasks as resulting from high arousal levels of the deprived smokers and low arousal levels of nondeprived smokers. They describe a number of studies that have shown arousal levels to interact with task difficulty in this way (e.g., Chiles 1958). However, smoking deprivation for 24 h reduces heart rate and hand tremor (Gilbert and Pope 1982). What is more, Williams (1980) used the smoking-based high arousal of nondeprived smokers as the means to explain their increased performance following smoking over deprived smokers on a simple letter-cancellation task during a sham smoking condition. Deprivation of smoking occurred for 24 h in the study of Kleinman, Vaughn, and Christ and this is considerably longer than the deprivation period in most studies including the typical morning of smoking deprivation used by Williams. Arousal may increase with longer periods of smoking deprivation.

Carter (1974) found no differences on a serial learning task when he compared nondeprived smokers with deprived smokers. The list consisted of 12 nonsense syllables and nondeprived smokers took two puffs during a 1-min rest period between learning-test trials and also smoked one cigarette before testing. Testing occurred again 7 d after initial learning and no significant differences in trials to relearning appeared between the deprived and nondeprived smokers. Neither group smoked prior to this retention test.

A recent extensive study of smoking and learning was reported by Peeke and Peeke (1984). In a series of experiments they explored the effects of smoking before learning on immediate recall, including smoking of cigarettes of different nicotine dose and smoking by people with different nicotine consumption levels. They also studied delayed recall as a function of this prelearning smoking and also as a function of smoking after learning which would relate to memory consolidation effects. The learning task was to remember lists of words presented via tape recorder. Typically, smoking before learning did increase memory for the words compared to the no smoking condition, but smoking after learning produced only a small insignificant improvement of memory or no improvement at all. Dose of nicotine in the prelearning cigarette was directly related to memory improvement although the highest dose used was only 1.38 mg of nicotine. The researchers expect that an inverted-U-shaped function describes the effects of nicotine on human learning (as it appears to with animals) and that stronger cigarettes would have produced less memory improvement. Smoking prior to learning was related to significantly improved recall as much as 24 h after smoking for one group of

smokers who normally consumed only small amounts of nicotine. Peeke and Peeke discuss that their consistent finding of small improvements in memory for words as a function of smoking before learning could reflect a normalization of deprived smokers instead of any facilitation of learning by smoking.

The absence of effects of smoking after learning found by Peeke and Peeke (1984) replicated results of Mangan and Golding (1983) who compared a no-smoking condition following paired-associate learning with conditions where low-nicotine (.8 mg), middle-nicotine (1.3 mg), and high-nicotine (2.0 mg) cigarettes were smoked after the paired-associates list was learned. No consistent differences were found for these different post-learning smoking groups for retention intervals of 30 min, 1 d, 1 week, and 1 mo, although the low-nicotine smokers showed more improvement over time than the other groups.

Learning and memory differences between smokers and nonsmokers: Hull (1924) not only compared learning of pairs of associations between geometric figures and nonsense syllables for smoking and "sham smoking" conditions, he also compared smokers and nonsmokers. His nonsmoker group was superior to the smoking group (the graph indicated approximately 11 trials to list learning for the nonsmokers compared to about 15 trials for smokers), but the difference did not quite reach statistical significance due to large differences among subjects within groups.

Kleinman, Vaughn, and Christ (1973) also compared nonsmokers with deprived and nondeprived smokers in an independent-groups design which looked at paired-associates learning for easy and difficult lists. They found the performance of nonsmokers to be highly similar to the nondeprived smokers who, as reported, were superior to deprived smokers for difficult lists, but inferior to deprived smokers for easy ones.

Weeks (1979) compared smokers and nonsmokers who were matched on alcohol consumption, age, and a number of other variables on their ability to learn associations between names and photographs. Ten min after the learning trials ended, the smokers achieved a score of 6.73 pictures correctly matched and nonsmokers achieved a score of 8.81 correct matches. This difference favoring nonsmokers over smokers was highly significant.

In their study of smoking and paired-associates learning, Mangan and Golding also compared nonsmokers with the smokers both on initial learning of the list of paired associates and on retention at the different periods following learning. Although there was no difference between these nonsmokers and smokers on initial learning, the nonsmokers showed much better

list recall than smokers at all retention periods. For example, 30 min following learning the nonsmokers required only an average of 1.67 trials to relearn the ten-pair list and this compared to an average of 3.59 trials for the smoker groups. Differences between nonsmokers and smokers in the number of trials to relearn the list at longer intervals were somewhat smaller, but still averaged more than one trial and always with fewer trials needed for the nonsmokers.

These large smoker-nonsmoker differences found by Mangan and Golding (1983) deserve further comment. Subjects were instructed not to rehearse the list during the 30-min period following acquisition, and were given material to read instead. It is possible that nonsmokers were less able to block out the paired-associate list during this period. Some of the results showing more focused attention during smoking (Knott 1978a, Andersson and Hockey 1977) might indicate the smokers were able to concentrate on the reading material better than the nonsmokers who may have had the recently learned list competing with the reading material and with rehearsal of the list occurring as another result. However, since the deprived smokers were inferior on recall to the nonsmokers and behaved very similarly to the smoking groups, this weakens this explanation of more focused attention (on the reading task) as a result of immediate smoking.

Another possible explanation of the improved retention of nonsmokers over smokers in the study of Mangan and Golding is the smoker or the nonsmoker group rehearsed the list during the period prior to first measurement of retention despite instructions not to and despite their report of compliance with these instructions. If it were the nonsmokers who rehearsed, such rehearsal helped, if it were the smokers, such rehearsal interfered (perhaps because of reinforcement of errors that crept in). Since smokers were much more apt not to follow instructions about returning for testing (see below), it would seem they would have been more apt to engage in rehearsal despite instructions not to do so.

Many subjects in the study by Mangan and Golding did not report for the retention session that occurred 1 mo following learning, despite prompting the day before. However, none of the 15 nonsmokers failed to report while there were 20 "quitters" among the 54 smokers and this was a highly significant difference. This differential quit rate for smokers and nonsmokers meshes with a number of results to be discussed in a subsequent chapter on possible differences in "character" between smokers and nonsmokers (see Chapter 10: "Smoking, abuse of other substances, delinquency, and driving accidents").

Problem solving

Carbon monoxide effects on problem solving: O'Donnell, Chikos, and Theodore (1971) found no effect of two levels of COHb (5.9 and 12.7 percent) on mental arithmetic performance, time estimation, or tracking tasks. Seppanen, Hakkinen, and Tenkku (1977) also found no effects of CO on fingertapping and on the Bourdon-Wiersma test (letter cancellation) even with COHb levels of 12 to 13 percent. These levels of COHb are well above COHb levels produced by smoking and it appears problem solving would not be degraded by the carbon monoxide content of cigarettes. However, Mihevic, Gliner, and Horvath (1983) recently reported a small deleterious effect from five percent COHb levels on performance of a secondary digit subtraction task that was performed simultaneously with a difficult tapping task that required accurate alternate positioning of a metal stylus on two separated narrow targets. However, this increase in reaction time for the secondary subtraction task may be an artifact since it is small and occurred only at intermediate levels of difficulty for the primary tapping task. Mihevic, Cliner, and Horvath found no difference in performance between air-breathing and CO-breathing conditions when the tapping task was made more difficult.

Immediate effects of smoking on problem solving: Hull (1924) found habitual smokers had higher speed on a mental arithmetic task in the smoking condition than in the control condition where they inhaled hot air instead of tobacco smoke. Nonsmokers also smoked, but they showed a small opposite effect, adding faster in the control condition than in the tobacco smoke condition.

Elgerot (1976) used Raven's Progressive Matrices test and compared smokers following a period of abstinence from smoking (15 h) and in another condition where they smoked before and during testing. Twenty percent more problems were solved in the abstinence condition than during the smoking condition. Similar significant differences favoring abstinence over smoking were found for another reasoning test and a mental arithmetic test. However, simple mental tasks of proofreading and perceptual speed (Bourdon Test) did not show a difference between smoking and abstinence conditions for these subjects. These results were similar to the results found by Williams (1980) where smoking interfered with a difficult memory-span task, but facilitated a simple letter-cancellation task.

MacDougall et al. (1983) looked at performance on a difficult computer game played either following an h of smoking deprivation or played immediately following a 3.5-min period of smoking. A significant 72-point increase in performance over base line performance was found for the deprived group. A

nearly significant 35-point decrease in performance from baseline occurred for the group that smoked. The increased performance of the deprived smokers over the nondeprived smokers was highly significant.

In the MacDougall et al. (1983) study, the baseline game and subsequent game for the deprived group were both played under conditions of smoking deprivation. Smoking deprivation preceded the baseline game for the smoking group, but their subsequent game was played following smoking. Things learned under smoking deprivation may not have all transferred to the smoking situation and state-specific learning effects such as those found by Peters and McGee (1982) could have been a factor in the substantially lower performance of nondeprived smokers compared to deprived smokers.

Carter (1974) compared nondeprived smokers with deprived smokers on a 20-trial letter-digit substitution task of about 40-min duration and found smokers performed significantly worse than deprived smokers on the last ten trials of the test. Smokers smoked a complete cigarette before the task and took two puffs during a 60-s interval between each of the 20 trials on the task. In a second session a week later, where neither group smoked during the task, the two groups of smokers did not differ. Although no explanation of the decrement is given by Carter, the cigarette before and 40 puffs during the 40-min task may have produced very high levels of nicotine which led to the decrement in performance either because of nausea or because the nicotine dosage was high enough to reduce arousal rather than increase arousal as is more typical of normal smoking doses.

Lyon et al. (1975) in a study of influences of alcohol and tobacco, found nondeprived smokers had shorter decision times than deprived smokers in a complex choice-reaction-time task involving light patterns presented on one or the other of two parallel vertical rows of four lights. Subjects pressed a button on the same side as the row of lights when an even number of lights were lighted and pressed a button on the other side from the lights when an odd number of lights were lighted. Decision times were nearly 1 s in duration for these complicated responses, but were more than ten percent shorter for nondeprived smokers compared to deprived smokers and nonsmokers. The nondeprived smoker superiority over deprived smokers was smaller and not significant in a condition where subjects drank a placebo, but it was highly significant when moderate amounts of alcohol were imbibed that boosted blood alcohols to the .05 percent range. Nonsmokers also were included in this research and nondeprived smokers were only superior to nonsmokers at higher levels of blood alcohol.

Problem solving differences between smokers and nonsmokers: Hull (1924) compared performance of smokers and nonsmokers on a mental arithmetic task and although nonsmokers were about 15 percent faster than smokers and made about 30 percent fewer errors than smokers, the differences between the two groups were not significant because of the large variation in performance of subjects within groups. Hull used the same smokers and nonsmokers for all of his tasks and this may account for the consistent, but nonsignificant group differences for different tests that favored nonsmokers.

Stevens (1976) found nonsmokers as a group performed significantly better than smokers on an anagram solution task and on two other problem-solving tasks. When the 50 smokers were divided into heavy smokers (greater than 13 cigarettes daily) and light smokers (less than 12 daily), it was found nonsmokers significantly outperformed heavy smokers on these same tests, but the differences between nonsmokers and light smokers were not statistically significant. Smokers were free to smoke during testing and it is not clear whether this inferiority of smokers to nonsmokers on these tasks was a long-term or short-term effect of smoking.

However, Walker et al. (1969) did not find differences between smokers and nonsmokers on Raven's Progressive Matrices Test. One major difference between this study and the study of Stevens (1976), was that Stevens tested males and Walker et al. tested females. However, it is not clear why sex should interact with smoking status on these problem-solving tasks. Another possibility is smoking was not allowed during testing and thus no immediate detrimental effects of smoking, such as those found by Elgerot (1976), existed in the Walker et al. study. Smoking during testing occurred for most, if not all, of the subjects who smoked in Stevens research.

A few studies have found smokers to be at an advantage over nonsmokers in cognitive tasks. Dicken and Bryson (1978) found among academic psychologists of advanced academic rank that smokers had significantly more books and articles published than nonsmokers. Warburton, Wesnes, and Revell (1984) found students who smoked at the University of Reading had higher academic averages than nonsmoking students. Both of these results suggest smoking can have a positive effect on productivity, perhaps through maintenance of arousal, or, more likely, through reduction or prevention of negative affect during unpleasant tasks of long duration.

Time estimation

Carbon monoxide effects on time estimation: A considerable number of studies of CO effects on time perception followed a report by Beard and Wertheim (1967) of substantial reduction in the ability to judge the duration of a 1-s tone following exposure to low levels of CO estimated to produce only 2.5 to 5 percent COHb. However, these numerous attempts generally failed to replicate this finding and levels of COHb of nearly 20 percent did not produce any effect on similar tasks (O'Donnell, Chikos, and Theodore 1971, Stewart et al. 1970, Stewart et al. 1973). More recently, Otto, Benigus, and Prah (1979) carefully replicated all conditions of the Beard and Wertheim experiment and also found no effect of low COHb levels on auditory time discrimination.

Immediate effects of smoking on time estimation: Ague' (1974) found rapid smoking increased estimates of the duration of a 5-s interval from presmoking estimates and these overestimates reached a maximum 10 min following smoking. The subject produced the 5-s interval by switching slides on a slide projector when he estimated 5 s to have elapsed. However, the differences in time estimation following smoking were not related to different nicotine levels of cigarettes including a no-nicotine cigarette. Differences from presmoking estimates also did not appear at the "slow" rate of smoking which more closely resembled normal smoking rates.

Leigh and Tong (1976) incorrectly report Ague' found nicotine effects on time production despite no difference between nicotine and nonnicotine smoking conditions. Leigh and Tong also incorrectly described the Ague' effects as underproduction although the "5-s" intervals produced following rapid smoking (of even nonnicotine cigarettes) by Ague's subjects were longer than the intervals produced prior to smoking.

In their own study, Leigh and Tong looked at the effects of smoking and alcohol consumption on time judgments with conditions of no alcohol and no cigarette, no alcohol and one cigarette, alcohol and no cigarette, and alcohol and one cigarette. Subjects were required to produce intervals of 1.5, 4, and 9 s by depressing a switch at the beginning and end of their estimated time interval. Results were somewhat mixed, but for production of short intervals (1.5 s) the effect of smoking without alcohol was to lead to underproduction relative to intervals produced prior to treatment. This suggests a speeding of an "internal clock" by smoking. Alcohol consumption tended to lead to overproduction suggesting a slowing of the "internal clock." Smoking and drinking alcohol reduced

this overproduction or even led to underproduction indicating that smoking was counteracting alcohol effects.

Tong, Booker, and Knott (1978) used a task where a light moved 50 cm from left to right, then disappeared at a position in front of the subject. The subject then was to activate a switch when he believed the light would have reached another position 50 cm to the right of its last position if the light had continued moving at the same speed. After each such estimation, the subject then was asked to reproduce the duration that the light was "on" with another switch. These time/position-estimation and time-interval-reproduction tasks were performed following smoking in one experimental session and at another session following a period of rest without smoking. Smoking led to shorter estimates of the time for the moving light to bridge the gap and also shorter estimates of the time the light was on. This "underproduction" of intervals following smoking supported the results of the earlier study of Leigh and Tong (1976) and is interpreted as a result of tobacco-based stimulation of brain-stem arousal systems.

Differences in time perception between smokers and nonsmokers: Koenig (1972) provided results that indicated nonsmokers are more "future oriented" than smokers and nonsmokers tie the present and future together more than nonsmokers. However, Koenig admitted his methodology was crude and his results not particularly strong. Koenig called for additional research to test the hypothesis that smokers are less oriented toward the future than nonsmokers.

Conclusions and military implications

Despite the typical facilitation of learning in rats by nicotine, most of the research indicates smoking detracts from learning in humans and research indicating improved long-term recall with smoking does not hold up under close scrutiny. However, the decrements in learning following smoking have usually been small, even when they were statistically significant. In the few studies where smoking facilitated learning, it was typically because nonsmoking smokers in comparison groups (or comparison conditions) were suffering unpleasant smoking-withdrawal symptoms that interfered with learning.

State-specific-learning effects may be real, however, and things learned following smoking may be poorly recalled in conditions where smoking is not possible. Soldiers who cannot smoke in actual combat probably should not smoke while training for combat.

Problem solving also appears to be hurt more often than helped when smokers are actively smoking during the tasks. What is more, it appears the more difficult the problem, the larger this negative effect of smoking on the problem-solving task. Presumably, the same mechanism could be involved in both the learning deficits and problem-solving deficits which occur with smoking since learning is a key part of the problem-solving process. Smoker-nonsmoker differences in problem solving show no particular trend with smokers at an advantage in some studies, at a disadvantage in others, and with no differences in yet another study.

Changes in time perception with smoking have not been studied sufficiently to form any conclusions, but it appears smoking has the immediate effect of making an interval of time appear longer than it is. Deprived smokers may experience the passage of time at a different rate than nondeprived smokers and nonsmokers. This could have implications for soldier performance in settings where smoking is impossible. Smokers would experience a change in their perception of the passage of time as a result of their deprivation while nonsmokers would not. Coordination of team activities might be influenced as a result.

Chapter 6

Effects of smoking on arousal and on ability to deal with stress, pain, and fear

Marshall (1947) reported many soldiers were too frightened to fire their weapons during their initial exposure to combat in World War II. Similar observations were made during the Korean War. If tobacco smoking produced a reliable reduction of fear and other emotions which strongly interfere with soldier performance in combat situations, or, if they allowed the soldier to function despite this fear (which may be the same thing), then it could be argued that smoking (or some other form of nicotine administration) should be encouraged for every soldier in combat situations. This would be particularly true if these benefits occurred without serious reductions in physical, sensory, or cognitive performance because of deleterious smoking effects.

This chapter will examine the research data that bear on effects of smoking and nicotine on arousal and on performance as it is influenced by these smoking-related changes in arousal. Even if smoking did improve performance in most soldiers, smoking's causal relationships to numerous diseases (see Chapter 9: "Smoking-disease relationships: Effects on productivity and absenteeism"), argue for a less-poisonous method for administering nicotine (the chemical most involved in relationships with arousal) than inhalation of tobacco smoke.

This chapter will first address one line of evidence that smoking affects arousal which is to review studies of the effects of stress on smoking behavior. These frequent increases in smoking during times of stress certainly reflect the perception of most smokers that smoking has a calming effect (see below). On the other hand, habitual smokers experience considerable stress when deprived of smoking (see Chapter 8: "The effects of tobacco deprivation") and use this experienced stress as one cue to smoke. It could be that stressors unrelated to nicotine deprivation produce smoking behavior simply because they mimic withdrawal stress. Whether such nonwithdrawal stressors are ameliorated by smoking or not is an empirical question.

Following the discussion of the effects of stress on smoking, research will be described that explored the effects of smoking on physiological arousal and on subjective arousal. A surprising and apparently contradictory set of findings exist in this area. As will be described, some physiological indicators such as heart rate almost invariably increase with

smoking, and this is true even when stressful tasks or other events already have produced substantial increases in heart rate prior to smoking. Others such as skin conductance usually decrease indicating a reduction in physiological arousal. One recent study (Golding and Mangan 1982b) showed cortical activation and skin conductance both increase when smoking occurs in conditions of low arousal and cortical activation and skin conductance both decrease following smoking in conditions of high arousal. The level of arousal or other aspects of the smoker and his situation may account for many of the apparent contradictions related to physiological changes with smoking.

The immediate effects of smoking on pain tolerance, on tolerance of other stressors, on fear, and on aggressiveness then are discussed. Such effects are particularly salient for soldier behavior. Finally, long-term effects of smoking on arousal are described. Whereas the short-term effects of smoking appear to be predominantly calming, evidence will be presented that long-term smoking appears to produce a more nervous or anxious individual.

Effects of stress on smoking behavior

Many people report one major reason they smoke is to reduce stress (Coan 1973, Ikard and Tomkins 1973, Matarazzo and Saslow 1960, McArthur, Waldron, and Dickinson 1958, Schneider and Houston 1970). Research also indicates stressful life (and death) situations frequently increase smoking. For example, smoking increased dramatically among the civilian population in England in World War II (Stepney 1980) and in Israel during the Yom Kippur War (Ben-Meir, 1977). Other research indicates that personnel in stressful professions such as soldiers (Ben-Meir), administrators (Caplan, Cobb, and French 1975), and nurses (Hillier 1981, Kirkby et al. 1976) smoke more than people in the general population. In line with these findings, Lindenthal, Myers, and Pepper (1972) reported smoking increased for individuals as a function of the number of life crises they experienced. Similarly, Billings and Moos (1983) reported heavy smokers had experienced more recent negative events than nonsmokers and they had fewer and less supportive social resources (e.g., fewer friends) than nonsmokers.

Stress also raises havoc with attempts to quit smoking. Shiffman (1982) found that negative affect was a major factor in the failure of smoking cessation treatments. People reporting several significant changes in their lives, such as a divorce, have more problems quitting smoking than people with fewer such changes (Benfari et al. 1982). The Yom Kipper War interrupted and totally obliterated the effects of two smoking cessation programs (Ben-Meir).

Studies have shown smoking tends to increase when people are placed in stressful experimental situations. For example, Schachter et al. (1977) showed the number of cigarettes smoked increased and the total number of puffs was significantly higher when subjects knew they awaited a session of painful electric shocks than when they knew they were waiting for a session of threshold-level shocks. In another study, Rose, Ananda, and Jarvik (1983) placed subjects into three situations, one anxiety provoking (stage fright produced by preparing a monologue for a videotaping session), one involving concentration on a boring digit-counting task, and one involving relaxation. Smoking of a cigarette was required at the halfway point in each condition and the dependent variables were the number of puffs and the volume of gases that were puffed per min. Both the anxiety and concentration conditions produced significantly more puffs and smoke volume than relaxation.

Cherek (1985) found the number of puffs, puff duration, and number of cigarettes smoked were related directly to the level of noise during 2-h sessions when industrial noise varied from 60 to 90 dB. The industrial noise was background to a task that required 100 successive lever presses to increment a counter and extinguish a light. Smoking was allowed throughout the session. Highly significant differences appeared between noise levels for number of puffs and puff durations by the subjects and the increase in number of cigarettes smoked with increasing noise approached significance. Subjects served as their own controls in separate sessions at different noise levels. A possible flaw in the study was that "All industrial noise levels were presented in an ascending sequence." Thus the changes in smoking behavior are confounded with session order. On the other hand, subjects repeated sessions at the base line (60 dB) session until the number of puffs taken per session stabilized and order effects were presumably minimal. Subjects also finished their repeated sessions with a final 60 dB session.

Using a within-subjects longitudinal design to study the effects of stress on smoking in real-world situations, Conway et al. (1981) showed tobacco consumption by Navy petty officers who supervised training of new recruits increased on high-stress days compared to low-stress days during the training cycle. Coffee consumption showed a parallel rise and fall associated with high- and low-stress periods, respectively. On the other hand, Kasl and Cobb (1980) did not find people who lost their jobs increased smoking as a result of this job loss. Reduced buying power associated with job loss would be expected to work against such an increase in tobacco consumption, however.

Acidic urine leads to faster elimination of nicotine from the body (Beckett, Rowland, and Triggs 1965). Research has shown stress acidifies the urine (Schachter et al. 1977), and it has been claimed increased nicotine elimination associated with increased urine acidity accounts for the stress-induced increase in smoking which serves to maintain "normal" nicotine levels among addicted smokers (Silverstein, Kozlowski, and Schachter 1977, Schachter, Silverstein, and Perlick 1977). Dobbs, Strickler, and Maxwell (1981) also found stress was related directly to both acidity of urine and to the number of cigarettes smoked. In their study, a relaxation training treatment led to less acidity of subjects' urine and less smoking in subjects despite exposure to a stressful situation. However, Cherek, Lowe, and Friedman (1981) acidified urine and failed to find an increase in cigarette smoking using highly sensitive measures of puff frequency and duration. Cherek, Mauroner, and Brauchi (1982) studied the effect of increasing the pH of urine (reducing urine acidity) and found statistically significant reductions in smoking behavior for five of seven subjects, but the differences were only about 10 to 15 percent. Benowitz and Jacob (1985) found acid loading increased daily nicotine intake by 18 percent, but their doses of ammonium chloride produced acidification of urine far beyond those associated with stress and they did not envision stress-related acidification producing any substantial alteration of nicotine elimination or any noticeable increase in nicotine intake. Given these later studies, it must be concluded that acid urine is not a determinant of stress-related smoking increments.

Not all research on the effects of stress on smoking has shown stress increases smoking. Glad and Adesso (1976), for example, did not find waiting to be evaluated on an oral presentation caused any increases in smoking behavior compared to another condition which involved waiting without the evaluation threat. This wait for evaluation was shown by subjective ratings of anxiety to be an effective anxiety-provoking situation. Glad and Adesso also looked at the effect on smoking rate of having either smoking or nonsmoking confederates present during the waiting periods. The failure of the stress condition to increase smoking ocurred despite a sharp increase in consumption of cigarettes when other people were present who were smoking.

Immediate effects of smoking on arousal

Nesbitt's Paradox

Smoking increases heart rate, blood pressure, and some other indices of physiological arousal (see below for refer-

ences) in much the same way that stressors such as threat of injury, sensory overload, and important examinations increase physiological arousal. Ironically, these very stressors often provoke smoking in many habitual smokers because smoking is perceived to reduce the stress (see above). This perceived calming of the emotions in the face of heightened physiological arousal has become known as Nesbitt's Paradox (Schachter 1973).

Gilbert (1979) has provided an extensive review of these "Paradoxical tranquilizing and emotion-reducing effects of nicotine." Although many studies have explored the physiological changes with smoking and other studies have looked at ratings of anxiety and emotion associated with smoking and smoking deprivation, few studies have studied systematically both sets of variables. What is more, the direction of the physiological changes themselves appear to depend on the situation. For example, in the research of Golding and Mangan (1982b), EEG and skin conductance changes following smoking indicated increased arousal in the condition of sensory isolation, and indicated a reduction of arousal during stressful loud white noise.

Vogel, Broverman, and Klaiber (1977) showed EEG responses that mimic the flicker rate of flickering photic stimulation differed in habitual smokers and nonsmokers with more "photic driving" in smokers. In addition, smoking a cigarette reduced photic driving in both groups. Adrenergic stimulants such as amphetamine and norepinephrine inhibit photic driving according to Vogel, Broverman, and Klaiber and smoking was seen by them to reduce photic driving by providing central adrenergic stimulation.

Vogel, Broverman, and Klaiber (1977) viewed the smoker-nonsmoker differences in photic driving as reflecting a relatively impaired central adrenergic functioning in the smokers that, in the absence of smoking or other central adrenergic stimulants, leads to depression, tension, anxiety, and agitation. Nesbitt's Paradox thus is explained by smoking producing adrenergic stimulation that relieves an unpleasant chronic adrenergic deficiency. In this explanation, increased heart rate, blood pressure, and decreased skin temperature are only byproducts of bringing the adrenergic system to a more normal and less tense, or, (still) paradoxically, less depressed, state. One possible mechanism for this would be to overcome the impaired cerebral circulation that has been shown to exist in smokers (Rogers et al. 1984a, 1984b).

Sedgwick et al. (1981) conducted a pilot study of some associations between behavioral stressors and physiological processes in 12 healthy men. They compared smoking with four other stressors: heat (1 h in a humid room at 42.5 degrees

Celsius), ingestion of fat (1 gr of rich cream per kg of body weight), exercise (three 18-min periods on a bicycle ergometer with a work load producing a 130-140 bpm heart rate) and psychological stressors (sexually explicit film, stage fright, and problem-solving tasks). Smoking produced the least physiological change of any of the five stressors on physiological variables such as epinephrine levels, norepinephrine levels, corticosteroid levels, heart rate, blood pressure, skin temperature, and cholesterol. In fact, smoking did not produce a significant change from resting levels for any of these physiological variables. This was in marked contrast to the effects of psychological stress and exercise. These data indicate smoking is not a particularly potent stressor relative to other environmental stressors and this makes Nesbitt's Paradox less paradoxical.

Other studies also have found physiological changes with smoking do not always appear. Fuller and Forrest (1977) measured skin conductance and muscle electrical activity and found smoking did not significantly change these measures in either relaxation or stress conditions (although there was a tendency to lower muscle tension following smoking). However, Fuller and Forrest did find increased heart rate with smoking (see below). The relatively small physiological stress associated with smoking also was shown by Erwin (1971) who measured heart rate changes during smoking on a psychiatric ward and used telemetry to record heart rate during normal "spontaneous" cigarette smoking. For the ten subjects observed, increases in heart rate during the course of the cigarette were less than 2 beats per min and in no case were they significantly different from zero. Nesbitt's Paradox would not exist for these subjects even if they did experience subjective calming effects with smoking since there was no paradoxical increase in physiological arousal.

However, although Sedgwick et al. (1981) and Erwin (1971) did not find increases in physiological responses with smoking, many other researchers have. This research is described in the next session.

Increases in arousal with smoking

Some of the most convincing evidence smoking is arousing already has been described in other chapters. The significant improvement of smoking on the ability to resolve flicker (e.g., Waller and Levander 1980) has been frequently replicated. Another line of evidence that smoking is a stimulant is that smoking often overcomes the depressant effects of alcohol on visual perception and other aspects of performance (e.g., Tong et al. 1974a). The increased ability to process rapidly

presented information, or, at least, the increased maintenance of such processing by smoking (Wesnes and Warburton 1978, Wesnes 1985), also implies a smoking and nicotine increase of arousal.

Heart rate and other cardiovascular activity: With few exceptions (e.g., Erwin 1971), dozens of studies have provided data indicating smoking (or an equivalent alternative administration of nicotine) reliably increases heart rate (e.g., Hull 1924), blood pressure (e.g., Frankenhaeuser et al. 1968), circulating levels of serum epinephrine and other adrenalcortical compounds (Frankenhaeuser et al. 1968), and constriction of peripheral blood vessels (e.g., Koch et al. 1980). In addition to this stimulation of the autonomic-nervous-system "end organs," smoking and nicotine typically produce effects on "brain" electrical activity recorded from the scalp which are similar to effects of stimulant drugs or stimulating events. These include increases in dominant alpha frequency (Knott and Venables 1977) and desynchronization of alpha (Conrin 1980). Increases in blood flow to the brain following smoking (Wennmalm 1982) also augur for cortical arousal increases.

Poulton (1977) showed smoking increased heart rate even when the heart rate already was increased by nervous anticipation of an examination. In fact, smoking a cigarette increased heart rate on the day of the examination to the same extent that it increased heart rate on other days when no examination was imminent. Fuller and Forrest (1977) also compared the effects of smoking on heart rate in conditions of high arousal (anxiety-eliciting film about industrial accidents) and low arousal (relaxation) and also found heart rate increased with smoking in both conditions. Golding and Mangan (1982b) showed smoking produced a comparable increase of heart rate in a sensory isolation setting where heart rate was low and in a stressful white noise environment where it already was elevated. However, Phelps and Gerdes (1979) reported smoking prior to a stressful task increased heart rate, but prevented further increases in heart rate when the task was presented. Only this latter isolated result would provide a heart-rate-related basis for any calming of the emotions by smoking.

MacDougall et al. (1983) found heart rate and blood pressure increased for subjects bot' as a function of smoking and as a function of playing a comp.ter game with stress-inducing instructions ("Try to get the highest possible score"). Only males were included in this study. When subjects both smoked and played the game, increases in blood pressure and heart rate were at least the sum of the increases found for groups receiving only one or the other "stressor." There was evidence that those subjects who showed large blood pressure changes as a result of the game also showed large

changes because of smoking. Such "hot" reactors might be particularly vulnerable to cardiovascular accidents.

Dembroski et al. (1985) conducted a similar study to that of MacDougall et al. (1983) with female subjects. They also found heart rate and blood pressure increased as a result of smoking or as a result of a stress-inducing computer game. For the subgroup of their female subjects who both smoked and played the game, the effects also were largely additive with some evidence of a synergistic effect where the total effect on heart rate and blood pressure from both sources of physiological arousal actually was greater than the sum of the separate sources when presented alone. The potential negative implications of such synergistic effects were discussed. Such large demands of the heart for oxygen from smoking and stress, plus the reduced oxygen available due to inhaled carbon monoxide, could lead to cardiovascular accidents.

Dembroski et al. (1985) found the stressor (video game) not only increased heart rate and blood pressure, it also produced large increases in ratings of the dimensions "tense," "nervous," "successful," and "physiologically aroused." The only effect of the smoking manipulation on these ratings was a small change (increase) on the dimension "involved." It is not clear why smoking, which produced large changes in heart rate and blood pressure comparable to the video-game task, did not produce large subjective changes on these arousal dimensions like the video-game task did. It could be that smokers become so habituated to their smoking-related heart rate and blood pressure increases that they do not notice them. Another possibility is the mechanism for these increases is physiologically much more direct than the increases associated with a stressor such as a video game. Cognitive mediation of a heart rate change may be necessary for a change in perception of "tense," "nervous," "aroused," etc.

Hatch, Bierner, and Fisher (1983) studied physiological changes associated with smoking during an extemporaneous speaking task that produced large heart rate increases during a preparation period and even larger increases in heart rate during the presentation. They studied heavy smokers who reported they smoked for relaxation in three conditions: no smoking, smoking of a low-nicotine cigarette (.09 mg nicotine), and smoking of a "high"-nicotine cigarette (1.25 mg). Ten smokers were included in each group. Comparisons were made on heart rate, blood pressure, scalp potentials, skin resistance, the State-Trait Anxiety Inventory, and ratings of motor behavior during the speech. None of the groups showed any significant differences on any of the dependent variables as a function of smoking or of the nicotine level of the cigarette. Significant heart-rate increases and significant changes in

many of the other physiological and self-rating variables occurred as a function of the different stages of the task (baseline, preparation, and presentation), but the different smoking conditions were reported not to have produced differences in the physiological variables, contrary to expectations generated by the studies of Nesbitt (1973), Gilbert and Hagen (1980) and numerous others. This failure to find additive effects of smoking and stress on heart rate is surprising given MacDougall et al. (1983), Dembroski et al. (1985), Poulton (1977), and other studies that have shown at least additivity and, in some instances, a synergistic effect, where stress and smoking lead to higher levels than occur for stress or smoking alone. One worries that analyses of covariance may have been used inappropriately by Hatch, Bierner, and Fisher to analyze their data. They did not find significant differences for baseline levels of physiologic activity for their different groups, but still covaried on these baseline levels and covaried despite the fact these baseline levels "... tended to be negatively correlated with deviations from baseline levels during both preparation and speech periods (italics added)." It is possible a great deal of careful data collection was wasted that presumably could have elucidated many of the issues related to concomitant physiological arousal and calming of the emotions following smoking.

"Brain" electrical activity: Changes in scalp electrical activity have been related to smoking with most studies showing changes, such as a higher frequency of alpha and more desynchronization of alpha, which indicate heightened arousal (Conrin 1980). Friedman and Meares (1980) found evoked responses to visual stimuli increased following smoking. Golding and Mangan (1982b) found alpha activity to decrease while smoking during sensory isolation and to increase during smoking in a stressful white noise environment. The implication was smoking increased cortical arousal when it was low and reduced arousal when it was high. This paralleled their results in the same experimental settings with skin conductance which are described below.

Muscle activity: Most muscular reflexes and other muscular activity are reduced following smoking (see below). However, the amplitude of finger tremor increased sharply at all frequencies following cigarette smoking (Lippold, Williams, and Wilson 1980, Shiffman et al. 1983) and these recent results using sophisticated frequency analyses support numerous studies that have found performance on tasks requiring steadiness of hand to be impaired by smoking (Hull 1924). Increased tremor, as increased heart rate, is one of the most reliable changes associated with smoking.

Fagerstrom and Gotestam (1977) found tonus of muscles at the back of the neck that are involved in holding the head upright increased with smoking of a high-nicotine cigarette (1.8 mg) and increased even more when it was smoked rapidly. The tonus of these muscles showed an insignificant decline during the smoking of a low-nicotine cigarette (.9 mg).

Russell, Epstein, and Erickson (1983) found muscle electrical activity recorded from the forehead initially increased above baseline levels following heavy smoking, but then decreased sharply during the smoking period (three cigarettes of the regular brand during a 20-min period) and tended to remain low during a subsequent "stressor" period of mental arithmetic. However, heart rate increased significantly from baseline during the smoking period (75.2 to 95.5 beats per minute) and remained above baseline during the mental arithmetic task. Skin conductance also increased significantly from baseline during the smoking period and remained above baseline during the mental arithmetic task. Thus, despite the drop in muscle electrical activity during the smoking period, the effects of smoking, in this study, appear to be primarily arousing.

Epstein et al. (1984) found the magnitude of muscle electrical activity (EMG) associated with a "maximal forearm flexor contraction" showed large differences between deprived and nondeprived smokers and also as a function of high-arousal and low-arousal situations. High arousal (a concurrent mental-arithmetic task) sharply reduced the magnitude of this contraction (or at least the EMG associated with it) compared to a relaxation condition. Smoking and the mental arithmetic task both sharply reduced the magnitude of this contraction in both high-arousal and low-arousal conditions for male smokers and in the low-arousal condition for female smokers. Although this was not the thrust of the research report, the implication is that both smoking and the mental arithmetic task were arousing. Surprisingly, smokers (especially when deprived) produced higher maximal contractions (as measured by EMG) than nonsmokers and this would imply that smokers were less aroused than nonsmokers. However, this does not jibe with most results comparing smokers and nonsmokers (see below). Given the large differences between deprived smokers and nondeprived smokers and the large differences between smokers and nonsmokers that were found in this study for magnitude of maximal contraction, it is unfortunate that other measures of the strength of contraction, other than EMG, were not included.

Skin conductance: Palmar sweating is another physiological function that frequently is directly associated with arousal and the effects of smoking on skin electrical conductance which measures this sweating have been investigated by a number of

researchers. Smoking-induced increases in skin conductance were found by Ague' (1974) and even nonnicotine cigarettes produced some increase. However, the increases occurred predominantly in afternoon sessions with little change in skin conductance associated with smoking in the morning. This indicated the effect was not particularly powerful. Golding and Mangan (1982b) found in sensory isolation the effect of smoking was to increase the level of skin conductance and even sham smoking produced a small increase. However, as will be described, most studies have shown reduced skin conduction following smoking. It appears baseline levels of stimulation and associated initial arousal levels are factors determining whether skin conductance is increased or decreased by smoking, unlike for heart rate, where elevation with smoking reliably occurs regardless of baseline levels.

Other physiological and behavioral processes: The increased duration of the spiral-after-effect following smoking found by Golding and Mangan (1982a) would indicate smoking increased arousal given earlier research on stimulant and depressant drug effects on this phenomenon. However, these same subjects showed smoking effects on skin conductance that indicated reduced arousal. One explanation offered to account for these apparent contradictions is smoking helps with inhibition of irrelevant or irritating stimuli, but reinforces those stimuli to which attention is directed.

Vestibular nystagmus refers to the involuntary eye movements that occur when a seated person is rapidly rotated in the dark. The eyes typically "follow" the unseen external world (slow phase) and then rapidly turn back (fast phase) to start this slow phase again. Tibbling and Henriksson (1968) showed smoking produced a dramatic change in vestibular nystagmus patterns indicating increased central arousal of the subjects. Smoking caused a much more rapid interruption of the slow "tracking" phase, nearly doubling the frequency and halving the amplitude of the eye movements. The effect began 4 to 10 s after smoking and lasted for several min following smoking. Subsequent research (Tibbling 1969) showed injected nicotine produced a similar effect and elevated carboxyhemoglobin from smoking nicotine-free cigarettes did not produce the effect nor did elevated levels of carbon dioxide.[1]

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[1] These changes in vestibular nystagmus with smoking augur for optokinetic nystagmus changes with smoking that might influence detection of targets from helicopters and other moving vehicles (See Chapter 12: "Needs for additional research on smoking and soldier performance").

Decreases in arousal with smoking

Smokers frequently claim smoking calms their emotions. For example, Ague' (1973) found "inner tension" was decreased following smoking with the decrease related to the amount of nicotine in the smoked cigarette. Nesbitt (1973) reported an unpublished study by Ikard, Green, and Horn (1968) that indicated 80 percent of their sample reported their smoking was relaxing and 75 percent disagreed with the statement that smoking a cigarette was stimulating. Linn and Stein (1985) looked at extremely heavy smokers and found most reasons given for smoking were related to stress reduction.

Pomerleau, Turk, and Fertig (1984) looked at anxiety associated with an anagram task (that already had been failed at least once) as a function of smoking a zero-nicotine or a regular cigarette prior to the anagram task. Anxiety was measured with the Spielberger State Anxiety Inventory immediately before and after smoking. Significantly larger drops in anxiety occurred following somking of the regular cigarette than following smoking of the zero-nicotine cigarette. Subjects were heavy smokers who had only been deprived of nicotine for 30 min prior to smoking the regular or zero-nicotine cigarettes.

Muscle activity: Smoking and nicotine from nonsmoking sources have effects on skeletal muscle activity that may indicate less arousal or more arousal, with the difference largely depending on the muscle system being investigated. The knee-jerk reflex was found to decrease after smoking (Domino and von Baumgarten 1969) with a corresponding decrease in phasic electromyographic activity of the associated muscle. This change began about 30 s after the first puff and continued throughout 4 min of smoking with the greatest decrease in knee-jerk reflex observed during the first min of smoking. The amplitude of the knee-jerk reflex remained depressed for a period of 30 to 120 s after smoking ceased, then gradually recovered. The rate of recovery was greatest during the first 10 min following the end of smoking, with reflex amplitude returning to control levels 25 min after the end of smoking in all but one of 35 cases. Heavy smokers tended to show the largest reduction in the reflex.

Clark and Rand (1968) also found a reduction in the knee-jerk reflex during and following smoking, with the knee-jerk returning to normal within 5 min of the end of the smoking period. Low-nicotine cigarettes did not produce the effect. Heavy tobacco users showed less effect than light smokers in the study of Clark and Rand, contrary to the results of Domino and von Baumgarten (1969) and it is not clear what produced this difference related to amount of tobacco use.

Similar to the knee-jerk decreases following smoking were the decreases in magnitude and number of jaw clenchings during aversive stimulation (exposure to very loud sounds) following either smoking or the intravenous and oral administration of nicotine (Hutchinson and Emley 1973). Similarly, Webster (1964) found a sharp reduction in spasticity following smoking of a cigarette by a patient with spastic paralysis.

Skin conductance: Gilbert and Hagen (1980) showed skin conductance during viewing of emotion-producing scenes was lower when a high-nicotine cigarette was smoked prior to viewing the scene than when a low-nicotine cigarette was smoked. This was true despite the heart rate being higher in the high-nicotine condition than in the low-nicotine condition. Mangan and Golding (1978) reported habituation of skin conductance responses to a series of tones was much faster after smoking a single cigarette than in a condition without smoking. Golding and Mangan (1982a) found smoking reduced the number of spontaneous fluctuations in skin conductance, speeded habituation of skin conductance responses to a repeated tone, and also reduced initial levels of skin conductance. These effects tended to be more pronounced for a 1.3 mg cigarette than for a .6 mg cigarette.

Similarly, Golding and Mangan (1982b) found skin conductance responses (SCRs) to bursts of loud aversive white noise were significantly smaller both during real smoking and during puffing on an unlighted cigarette than in a control condition where no cigarette was involved. Differences in SCRs were not found between real smoking and sham smoking.

Boyd and Maltzman (1984) found skin conductance responses during auditory choice reaction-time tasks (with responses made via foot pedals) were significantly smaller following smoking than following abstinence. However, spontaneous SCRs during rest were larger following smoking than following abstinence, at least for the group of "high craving" smokers who were most bothered when not smoking. This would appear to parallel the findings of Golding and Mangan (1982b) who found reduced skin conductance responses after smoking during exposure to aversive noise and increased responses after smoking in sensory isolation.

Boyd and Maltzman (1984) found high craving smokers showed differences in skin conductance responses for the two hands following smoking compared to responses during smoking abstinence. Smoking increased right-hand SCR magnitudes compared to abstinence. They suggest the subjective calming during smoking may be related to increased activity in the right hemisphere that balances a relatively overactive left hemisphere which other research (Tucker 1981) has indicated may be a factor in

obsessive compulsive states and anxiety. Boyd and Maltzman admit this explanation of the subjective calming with smoking is speculative. They also do not provide an explanation of why increased hemisphere activity would influence skin conductance of the hand on the same side as the hemisphere given that hands are normally controlled by the contralateral hemisphere.

"Brain" electrical activity: Golding and Mangan (1982b) found smoking increased alpha activity recorded from the scalp during a stressful white noise session. Increases in alpha are typically a result of a reduction of arousal. Other evidence of a depressant effect of smoking on cortical activity comes from Friedman, Horvath, and Meares (1974) who showed subjects much more quickly habituated (did not show EEG changes) to a repeated tone after smoking than in conditions without smoking or with smoking of a nonnicotine cigarette. A later study of evoked cortical potentials (Friedman and Meares 1980) showed a similar reduction with smoking of EEG responses for auditory stimuli. However, as mentioned, cortical responses to visual stimuli were increased by smoking in the same subjects. The results suggest smoking reduces arousal of the auditory system, but increases arousal of the visual system. Pupillary changes were discounted by Friedman and Meares as an explanation of the higher responses to visual stimuli. However, smoking does increase the diameter of the pupil (Roberts and Adams 1969), which would increase the amount of light entering the eye and effectively increase the intensity of these visual stimuli. Intensity does increase the evoked response as is even shown by the increased levels of response as intensities increased in the study by Friedman and Meares. Increased macular blood flow (Robinson, Petrig, and Riva 1985) following smoking also may contribute to the differential effect found by Friedman and Meares for auditory and visual evoked potentials.

The contingent negative variation (CNV) is a shift in the level of scalp-recorded electrical activity that occurs between a warning stimulus and a response stimulus. Ashton et al. (1974) found the stimulant caffeine increased the magnitude of the CNV, the depressant nitrazepam reduced the CNV, and smoking produced increases in the CNV for some subjects and decreases in the CNV for others. The changes, though in different directions, were significant for individual subjects. It was suspected different CNV changes for different subjects were the result of different doses of nicotine related to more or less puffing of the individual subject. In a later study, Ashton et al. (1980) presented nicotine intravenously and found low doses increased the CNV and large doses depressed it and this supported the earlier interpretation of the different effects of smoking on the CNV for different subjects.

Other physiological processes and behaviors: Hartley (1973) found smoking caused changes in the way people observed three different sources of signals which had different probabilities of the signal appearing. Previous research using this task has shown increased arousal (e.g., from a noisy environment) typically caused people to increase sampling of the higher probability source and decreased arousal (e.g., following sleep deprivation) caused people to increase sampling of the lower probability source. Hartley found smoking either one or two cigarettes before this task had the effect of causing subjects to select from the lower probability source, suggesting smoking caused a reduction in arousal.

Tobin, Schneider, and Sackner (1982) found smoking produced a slowing of breathing in some smokers and subsequent research (Tobin, Jenouri, and Sackner 1982) indicated the body's natural opiates or endorphins were involved. This depression of the respiratory center and other effects of endorphin production may be another key to the frequently reported relaxation following smoking, despite the heightened heart rate and other increases in physiological responses.

Arousal increase or arousal decrease with smoking?

On balance, an arousal decrease with smoking appears to be the more dominant finding with more physiological processes being depressed than activated by smoking. Heart rate changes with smoking were almost invariably increases, but the increases in heart rate with smoking do not appear to produce subjective arousal changes or to be accompanied by subjective arousal changes such as those which occur when heart rate is increased by a difficult task. The smoking influences on subjective feelings of arousal appear to be primarily relaxing. The exception may be the situation of sensory isolation where arousal is at very low levels and smoking is a major stimulus event. However, despite reduced physiological arousal and reduced subjective arousal with smoking, the higher critical flicker frequencies following smoking, smoking's countering of depressive effects of alcohol, and the improved information processing and vigilance following smoking, all indicate the brain of the smoker usually is working more effectively following smoking. Nesbitt's Paradox which relates to heightened physiological arousal and subjective calming appears also to include increased cortical efficiency and subjective calming.

Smoking and tolerance of pain

If smoking reduced arousal or emotionality, this might increase a person's ability to tolerate pain. However, smoking

could be analgesic even without a change in arousal emotionality. Nesbitt (1973) defined emotionality as low tolerance for painful electric shocks and found deprived smokers were high in "emotionality" by this definition since they stopped a series of increasing shocks at an early point in the sequence. Smoking of cigarettes reduced "emotionality," i.e., increased pain tolerance in these formerly deprived smokers. High-nicotine cigarettes increased pain tolerance more than low-nicotine cigarettes and heart rate increases with smoking were positively correlated with increases in pain tolerance. Nesbitt also found nonsmokers did not tolerate shocks as intense as those endured by nondeprived smokers and concluded smoking did more for pain tolerance than just to eliminate nicotine-withdrawal based arousal that reduced pain tolerance. However, smoking nonsmokers did not tolerate more pain in the study by Nesbitt. This was explained by the dizziness and upset stomachs smoking produced, indicating an already higher "emotionality" for the nonsmokers.

However, Silverstein (1982) found no difference in pain tolerance between smokers and nonsmokers despite replicating most of the other results of Nesbitt (1973). Silverstein interpreted his and the Nesbitt results as reflecting a reduction of a smoking-deprivation effect with smokers "seeking nicotine rather than relaxation" and thus their increased pain tolerance following smoking was not a true reduction of emotions with smoking. However, smokers of high-nicotine cigarettes did require higher shock levels than nonsmokers before they reported shocks were painful. As Silverstein notes, "It may be that at the very low levels of stress represented by the pain threshold, nicotine exerts a calming effect."

Shiffman and Jarvik (1984) attempted to replicate the results of Nesbitt and Silverstein. Shock intensity was similar, but their results differed in many respects from those of both previous studies. Nearly all subjects were able to endure the whole series of shocks so the endurance threshold that was the major dependent variable in the other two studies could not be used. For the (lower) threshold of pain, which was reached by all subjects, smoking and sham smoking produced no differences. An anxiety scale completed after each session also showed no differences between the smoking and sham smoking conditions. Finally, where Nesbitt found a positive correlation between heart rate and ability to endure pain, Shiffman and Jarvik found a significant negative correlation indicating high heart rates and low pain thresholds went together. Nesbitt's Paradox is absent since high physiological arousal accompanies high emotionality. Several differences between this more recent study and the earlier two existed. One key one, which Shiffman and Jarvik emphasize, is the more automated

procedure in their study which reduced subject-experimenter interactions and the possibility of experimenter-expectancy effects.

Schalling and Waller (1980) provided little description of the methodology of their study, but reported male smokers increased their discrimination of the magnitude of different painful electrocutaneous stimuli and also increased their tolerance for such painful stimuli after smoking.

Waller et al. (1983) attempted to replicate the Nesbitt (1973) study and included a condition where heart rate changes with smoking were blocked with a beta-blocking drug. Smoking with or without beta blockade did not change either pain thresholds or pain tolerance levels.

However, Pomerleau, Turk, and Fertig (1984) were able to replicate the Nesbitt and Silverstein (1982) effect of nicotine on smokers, although instead of looking at pain and pain-tolerance thresholds for electric shocks of increasing intensity, they looked at the time subjects could keep their hand and forearm in ice water (pain-tolerance threshold). Subjects also were to indicate when this first became painful (pain threshold). A within-subjects design was used with this "cold-pressor" test immediately preceded by a regular cigarette on one day and a zero-nicotine cigarette on another. Five heavy smokers participated in the experiment and each had smoked his regular cigarette 30 min prior to the cold-pressor test, making him "minimally-deprived" of nicotine. Pain thresholds were elevated significantly following the regular cigarette compared to the zero-nicotine cigarette with an average 77 percent increase in time before pain was indicated for the regular cigarette compared to the zero-nicotine cigarette. Pain tolerance thresholds did not differ significantly, but were higher for four of the five subjects following the regular cigarette with the fifth subject maintaining his arm in the ice water for the full 5 min following both cigarettes. Since smokers had been deprived of nicotine only 30 min, Pomerleau, Turk, and Fertig saw the effect as being a nicotine-based increase in pain tolerance and not a reduction of withdrawal symptoms. However, these subjects averaged nearly a cigarette every 30 min in their regular smoking and this conclusion is debatable. Also the half-life of nicotine is only 12 to 15 min (Sepkovic et al. 1983). It is unfortunate nonsmokers were not compared to deprived and nondeprived smokers on their cold-pressor test.

Perceived exertion may be thought of as one form of pain and perceived exertion also appears to show changes with smoking. Morton and Holmik (1985) obtained ratings of perceived exertion once per min during treadmill testing in a

study of the effects of smoking on maximal oxygen consumption in elite team athletes who were smokers and nonsmokers. No differences appeared between smoking and nonsmoking groups in perceived exertion, but perceived exertion was rated significantly less at certain periods of testing following the smoking of two cigarettes for both smokers and nonsmokers. This reduction in perceived exertion following smoking could allow more pain tolerance and harder efforts following smoking because of the antinoceptive effects of nicotine and might counter COHb or other smoking-related performance limiters.

Smoking and tolerance of fear and other stressors

Research on animals has shown nicotine reduces fear leading to improved avoidance of electric shocks (e.g., Hall and Morrison 1973) and also leading to increased exploration in novel environments (Battig 1981). Although many of the observations of increased smoking in wartime and in other stressful situations which already have been described can be interpreted as attempts at fear-reduction through smoking, no scientific studies were found which looked at smoking and fear in humans. Ethical considerations appropriately restrict the imposition of fear-arousing stimuli on humans and this undoubtedly is one reason no such studies exist. In addition, collection of research data is not a prime consideration in natural disasters, combat situations, and other dangerous situations where fear is endemic. A partial way of meeting the need for research on the effects of smoking on fear in humans would be to obtain information on the effects of smoking from people such as combat veterans who have smoked or observed smoking in fearful situations (see Chapter 12: "Needs for additional research on smoking and soldier performance"). Another (admittedly "far-out") possibility for obtaining data on smoking and fear would be to find television camera news footage showing men in combat, civilian hostages, or people exposed to other dangerous situations that illustrates changes in performance or changes in facial expressions or other outward signs of fear in smokers following the lighting and smoking of cigarettes.

Ague' (1973) found smokers smoking high-nicotine cigarettes gave higher ratings of the adjectives "refreshed," "pleased," "lighthearted," "relaxed," "happy," and "joyful" when describing their mood than smokers who smoked cigarettes with low nicotine or no nicotine. These smokers had gone without cigarettes for 8 h prior to smoking and these differences in ratings undoubtedly reflect reduction of unpleasant smoking-withdrawal effects.

Heimstra (1973) reported measures of mood made before and

after a series of five arduous vigilance and tracking tasks. Smokers allowed to smoke during these tasks typically reported fewer and smaller shifts in mood than deprived smokers and nonsmokers. The many studies that have shown nondeprived smokers to outperform deprived smokers and at times to even outperform nonsmokers in long vigilance, reaction-time, and other tasks, probably reflect the better mood of nondeprived smokers in these situations as much as any heightened capacity for performance.

Aggressiveness changes with smoking and nicotine injection

In both man and animals, smoking and nicotine injections have led consistently to a reduction of aggressiveness. Rodgers (1979) and Driscoll and Baettig (1981) showed small doses of injected nicotine reduced shock-induced fighting in rats while not altering shock sensitivity or depressing activity. Hutchinson and Emley (1973) made a similar finding that nicotine reduced biting induced by electric shock in monkeys and also reduced jaw clenchings induced by aversively loud tones presented to humans.

Cherek (1981) studied the effect of smoking on human aggression and found aggressive responses in each of eight smoking subjects decreased if experimental sessions were preceded by smoking of two cigarettes. Subjects performed a reaction time task that accumulated money. Random subtractions of money from their total were attributed to another "subject" who presumably was pressing a button that took the money away from him. The subject could press a button and presumably take money from this person, as well. Aggressiveness was defined as the number of such money-subtracting responses. These responses were much more apt to occur when subjects did not smoke in the 30-min period prior to the experimental session than when smoking of two cigarettes occurred prior to the session. The effect was related to nicotine level of the cigarettes. In seven of eight subjects there was less aggressiveness when the cigarettes were high-nicotine (2.19 mg nicotine) than when they were low-nicotine (.42 mg nicotine).

Schechter and Rand (1974) used the "Buss aggression-machine" in which subjects punish their "partners" during a learning task when they make errors to "help them learn faster," and where the number and duration of these shocks is the measure of aggressiveness. They found smokers had higher aggression scores when they were deprived of cigarettes than when they smoked. They also compared nonsmokers with nondeprived smokers and although nonsmokers had a considerably higher aggressiveness score than nondeprived smokers, this difference between the two groups apparently was not significant.

Long-term effects of smoking on arousal

Coan (1973), Schneider and Houston (1970), Matarazzo and Saslow (1960), and Williams, Hudson, and Redd (1982) reported smokers to have higher anxiety scores than nonsmokers. This generally was interpreted as reflecting increased smoking by anxious persons, but the reverse interpretation, smoking increases anxiety, also may be valid as Russell (1971) suggested. Spielberger and Jacobs (1982) found females to be higher in anxiety, but male smokers had lower anxiety scores than nonsmokers. Blackburn et al. (1960) and Thomas (1960) reported smokers generally have higher heart rates than nonsmokers and Nesbitt (1973) also found a correlation between number of pack-yr (yr of smoking multiplied by number of packs daily) and basal pulse rate for the 29 smokers in his experiment. Knott (1980) measured skin conductance of nonsmokers and smokers who were deprived for 13 h and found skin conductance was lower and dropped more quickly during rest periods for nonsmokers than for smokers. However, Gofin, Kark, and Friedlander (1982) found blood pressure to be lower in adult male smokers and pulse rate only slightly higher.

In a community study of 1,209 men and women in New Zealand, Waal-Manning and de Hamel (1978) found nonsmokers, light smokers, moderate smokers, and heavy smokers showed progressively larger somatic anxiety scores. Parallel increases in somatic anxiety with rate of smoking were found for men and women. Williams, Hudson, and Redd (1982) reported items dealing with somatic symptoms such as gastrointestinal problems, body temperature problems, shortness of breath, etc., were more apt to be reported by smokers than nonsmokers.

Lawton and Phillips (1956) reported heavy smokers were much more apt to report they were considered by others to be a nervous person than moderate smokers. Their interpretation was nervousness caused the smoking, but it is possible heavy smoking produced the nervousness, instead. Moodie (1957) found no association between smokers and nonsmokers on reported nervousness, but did find a significant association between amount of smoking of cigarette smokers and nervousness. Persons smoking more than 18 cigarettes daily being more apt to judge themselves as nervous than persons smoking less.

Tagliacozzo (1982) found nurses who smoked reported higher levels of strain, tension and conflict in their working situation than their counterparts who did not smoke. These different perceptions of smokers and nonsmokers undoubtedly reflect more on the nurses than any differences in their actual work situation and are included in this section on smoker nonsmoker-differences in arousal. The effects of this stressful work environment on nurse smoking rates were described earlier.

Knott (1984) found skin conductance levels and skin conductance responses of smokers were higher than for nonsmokers with the effect primarily related to female smokers. He describes this as supporting "... the contention that smokers are characterized by a state of relative hyperarousal and hypersensitivity to stimulus input." However, these were heavy smokers and they had been deprived of tobacco for several h and this might reflect a smoking deprivation effect as much as or more than a smoker-nonsmoker difference.

Fagerstrom and Gotestam (1977) reported an observation which was not backed up by any data, but which corresponds to the author's personal experience as a former smoker. They describe, "Another interesting observation is that some very tense and hyperactive smokers have changed dramatically to much more relaxed and quiet individuals when they stopped smoking." The direction of the association between smoking and anxiety or between smoking and physiological activation generally is assumed by researchers to be such that anxiety differences produce the smoking differences. What is more, data collected on children prior to starting smoking have shown some differences on such variables already exist. However, it is probable the Fagerstrom and Gotestam observation above also is valid and at least some of the smoker-nonsmoker differences reflect effects of long-term smoking with smoking increasing anxiety and somatic symptoms (Russell 1971).

However, not all studies have found smokers to be higher in arousal than nonsmokers. Ek et al. (1977) conducted an extensive study of Swedish soldiers, 110 of whom were smokers and 19 of whom were nonsmokers. They ranged in age from 20 to 24. Relatively few differences were found between smokers and nonsmokers. Serum iron was higher for nonsmokers, serum zinc was higher for smokers, and the rating of "concentration" was higher for nonsmokers. However, for the complex of variables most closely associated with stress, such as serum epinephrine, norepinephrine, and T_4 iodine, and rated "tension," "distress," and "alertness," no significant differences appeared between smokers and nonsmokers. Smokers and nonsmokers also showed few differences when viewing a "stressor movie."

However, the failure of Ek et al. to show smoker-nonsmoker differences on arousal-related variables does not preclude a long-term smoking increase of anxiety and arousal. Their failure to find differences may reflect the young age of these men, or, more correctly, their short smoking histories. With increased exposure to smoking, these soldiers who smoked might demonstrate differences from nonsmokers similar to those reported by Waal-Manning and de Hamel (1978) and others which examined large age ranges. It would be particularly interest

ing to assess anxiety differences between smoking and nonsmoking subjects of the Ek et al. study now.

Conclusions and military implications

The short-term calming effects of smoking suggest some form of nicotine administration could improve soldier performance in those situations where soldiers are so frightened or otherwise aroused they "freeze" and cannot even fire their weapons. Should research confirm that nicotine can improve performance under these circumstances, smoking may not be the best battlefield solution, particularly at night. Nicotine tablets, nicotine aerosols, or any other drug-based calming also is probably fraught with problems, however, and research is needed to identify possible health, addiction, and other problems that could result if these forms of nicotine were used to alleviate stress. Still, the life and death consequences of an overaroused soldier for himself and his unit might justify nicotine or other drug interventions. These consequences surely justify the research that is needed to examine these possible means for countering anxiety-induced performance deficits.

The smoking-induced decrease of reflexes and other major motor activity suggests major muscle movement (e.g., flinching) may be reduced in shooting following smoking. The smoking-induced increase in tremor of the hands suggests fine control of weapon pointing would be impaired following smoking. A possible net outcome would be good shooters would reduce their performance and poor shooters (at least, those who flinch) would improve their performance after smoking. Research on the effects of shooting on smoking is recommended in Chapter 12: "Needs for additional research on smoking and soldier performance" at the end of this report.

The short-term calming effects of smoking appear to lead to long-term higher levels of arousal and nervousness in smokers. More research is needed to establish this probable effect, but even without additional research, this strong possibility could be exploited in smoking cessation programs. The large number of smokers who smoke to calm themselves should be particularly interested in "not smoking" in order to really calm themselves.

Chapter 7

Smoking-induced hormonal changes

Hormones have an immense influence on growth, sexual maturation, mood, arousal, behavior, and even on mental processes (Sawin 1969). Smoking reliably stimulates production of nearly a dozen hormones. Production of other hormones, including the major sex hormones (testosterone in males and estrogen in females), appears to be reduced by long-term smoking, although conflicting results exist for testosterone. Many of these hormonal changes occur within s or min of inhalation of tobacco smoke and most appear to be related directly to the nicotine content of cigarettes. In some instances, the acute effect of smoking is an increase in hormone production, but the long-term effect is a decrease (see results for prolactin below).

Many of the reliable effects of smoking on heart rate, blood pressure, muscle tremor, etc., are preceded directly and caused by rapid smoking-related changes in hormonal production (Burn 1960, Carruthers 1976). Also, it is probable that hormonal changes resulting from smoking may provide much of the "reinforcement" that causes people to continue to smoke in the face of indisputable deleterious effects on health (Chernick 1983, Karras and Kane 1980). However, this separate section on hormonal response is included since knowledge of smoking-produced changes in hormones, combined with our knowledge of the effects of these hormones, may help us to identify effects or potential effects of smoking on soldier performance where the performance data currently do not exist.

Testosterone

Testosterone is the major gonadal hormone in males and many of its effects on health and behavior are fairly well understood. Although there are contradictory findings related to both long-term and immediate effects of smoking on testosterone production, testosterone production may be reduced in habitual smokers. This was clearly shown in research of Shaarawy and Mahmoud (1982) who found serum testosterone levels in smokers were only one-half the testosterone levels in nonsmokers. Urinary 17-oxosteroids, which are metabolic by-products of testosterone, also were sharply decreased in smokers relative to nonsmokers which suggests it was not just more rapid clearance of testosterone by smokers than nonsmokers which caused the difference. In addition to the testosterone differences, sperm count and sperm motility were significantly lower for smokers than for nonsmokers. Unlike infertile

subjects in some studies of gonadal hormones, the 25 smokers and 20 nonsmokers selected for this research by Shaarawy and Mahmoud all were free of hormonal or fertility problems. Each had fathered at least two children including one within 2 y of the study.

This result of Shaarawy and Mahmoud supported earlier research by Briggs (1973) which showed plasma testosterone levels were significantly lower in heavy smokers (5.15 ng/ml) compared to nonsmokers (7.47 ng/ml). Briggs also found abstaining from smoking for 1 week caused a significant rise of 1.65 ng/ml in plasma testosterone levels of the six smokers in his study. Mellstrom et al. (1982) studied 70-yr-old males and found smokers had a higher ratio of estrogen to testosterone than nonsmokers and this result also may provide support for the results of Briggs and Shaarawy and Mahmoud. Still further support for Shaarawy and Mahmoud and other studies showing a reduction of testosterone with smoking comes from a study by Mittler, Pogach, and Ertel (1983) who measured testosterone levels of beagles that inhaled the smoke from 12 cigarettes daily for about 2 yr with the smoking of the dogs controlled by a machine that duplicated standard human-puff profiles. They found serum testosterone levels were reduced by 54 percent from control levels by smoking. Briggs, Mellstrom et al., and Shaarawy and Mahmoud appear to have identified a reliable longer-term diminution of testosterone production in man as a result of smoking and if one stopped in a literature survey in 1983 (as a preliminary version of this review did), the conclusion would be that smoking definitely reduced testosterone production.

However, Handelsman et al. (1984) studied the differences between smokers and nonsmokers in testicular function among potential sperm donors. Smokers were not found to have lower testosterone levels than nonsmokers, although there was significantly lower sperm output for smokers (181 million per ml) than for nonsmokers (316 million per ml). Smokers also demonstrated significantly lower sperm motility than nonsmokers. Tsitouras, Martin, and Harman (1982) also found no significant difference between smokers and nonsmokers in serum testosterone levels in their study of 183 healthy men aged between 60 and 79. The failure of Handelsman et al. and Tsitouras, Martin, and Harman to find testosterone differences between smokers and nonsmokers leads one to interpret the lower testosterone levels for smokers found by Briggs and Shaarawy and Mahmoud with some caution.

One becomes somewhat skeptical about the lower testosterone level findings for smokers given even more recent studies of this relationship. Deslypere and Vermeulen (1984) found significantly higher plasma testosterone levels in smoking men

than in nonsmoking men. This significant difference held in each of three age groups: 20-39, 40-59, and 60-80, with the average difference for all age groups a substantial 131 ng/dl. Deslypere and Vermeulen have two figures presented over the wrong captions in their report and one wonders if they are reporting their results for smokers and nonsmokers correctly. On the other hand, they discuss the discrepancies between their work and the results of Briggs (1973), Shaarawy and Mahmoud (1982), and Tsitouras, Martin, and Harman (1982) so they would hardly have been apt to mix up the data from smokers and nonsmokers. Deslypere and Vermeulen also found significant declines in testosterone with increasing age, but, unlike the higher testosterone for smokers, these were expected.

Other support for higher testosterone levels in smokers comes from Andersen, Semczuk, and Tabor (1984) who measured testosterone in infertile men and found significantly higher levels of testosterone in smokers than in nonsmokers. Plasma testosterone levels averaged 18.9 nmol/l for nonsmokers and 21.3 nmol/l for smokers. They also were aware of the contrast of their results with those of Shaarawy and Mahmoud (1982) and suggested the difference may be related to their infertile sample which contrasted with the fertile sample of Shaarawy and Mahmoud.[1] Another possibility they suggested was that the weight difference between smokers and nonsmokers might account for part or all of the difference since obese males have been shown to have lower plasma testosterone levels than nonobese males (Amatruda et al. 1978). However, the difference in weight between the nonsmokers and smokers was only 3.9 kg and the variance of weight for nonsmokers did not differ from smokers suggesting no large amount of obesity in the nonsmokers. In the Amatruda et al. study that showed low testosterone to be related to obesity, the obese men weighed from 176 to 200 percent of ideal body weight.

These contradictory results related to differences between smokers and nonsmokers in levels of testosterone indicate a need for additional research to identify the circumstances when testosterone levels are positively and negatively associated with long-term smoking or to determine which relationship holds if some of the previous research is invalid.

Shorter-term effects of smoking on testosterone also are ambiguous. Persky et al. (1977), in a study of the effects of alcohol and smoking on aggression and testosterone in chronic

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[1] There are obvious errors in the text of Andersen, Semczuk, and Tabor (1984) where "infertile" is substituted for "fertile" on pages 392 and 395.

alcoholics, noted a small but significant positive correlation ($r=.40$) between smoking and testosterone level over a 1-week period.[2] These were men who were consuming alcohol and who generally showed a decrease in testosterone during the week compared to testosterone levels during a previous nondrinking week. However, the more the man smoked, the smaller was the reduction of testosterone during the week of resumed drinking. Mattison (1982) reported results of a study by Halawa and Mazurek (1977) who demonstrated smoking "may elevate testosterone." It is not clear from the Mattison summary whether this possible increase was a long-term or acute effect of smoking.

Dotson, Robertson, and Tuchfeld (1975) found changes in testosterone levels were positively correlated ($r=.24$) with the amount smoked during an evening "party" experiment. Measures were made immediately before drinking began and again after the 3-h party. Alcohol consumption was positively correlated with testosterone changes, as well ($r=.26$). The correlation of alcohol consumption with cigarette consumption was not significant ($r=.11$) suggesting both smoking and alcohol had independent influences on testosterone production, metabolism, and/or clearance. The correlation between smoking and testosterone change was small, but with 91 subjects (six different parties) was significant.

Both Persky et al. (1977) and Dotson, Robertson, and Tuchfeld (1975) found what appears to be a short-term increase of testosterone production resulting from smoking. However, another study that examined immediate effects of heavy smoking (eight 2.5 mg nicotine cigarettes in 2-h) on testosterone levels showed neither an increase or decrease (Winternitz and Quillen 1977). It could be the Persky et al. and Dotson, Robertson, and Tuchfeld results were influenced by the alcohol consumption which occurred along with smoking in each study.

Assuming smoking-induced suppression of testosterone were a reality, or, as more recent evidence may indicate, that smoking increases testosterone production, either change could have important effects on behavior, health, and other aspects of human existence. Testosterone has important roles in behavior of humans, as well as other primates, and other animals. In humans, it has been related to aggressive behavior (Kreuz and Rose 1972, Ehrenkranz, Bliss, and Sheard 1974), social status (Mazur 1976), sensation-seeking (Daitzman et al. 1978, Daitzman and Zuckerman 1980), mood (Klaiber et al. 1976,

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[2] Mattison (1982) reviewed literature related to smoking and fertility and incorrectly reported Persky et al. (1977) had found a "significant inverse correlation between cigarette consumption and testosterone levels."

Prange et al. 1977), and sexual arousal (Rubin et al. 1979).

Rada, Kellner, and Winslow (1976) reviewed testosterone and aggressive behavior and found somewhat conflicting results. However, more studies showed a direct relationship between testosterone levels and human aggressive behavior than did not. Aggressive behavior and combat are more or less synonymous. If testosterone levels of soldiers who are habitual smokers are reduced as the Shaarawy and Mahmoud (1982) results would suggest, the expected consequent reduction of aggressive behavior associated with suppression of testosterone would be expected to impair combat performance. If testosterone and smoking are positively correlated, as Andersen, Semczuk, and Tabor (1984) and Deslypere and Vermeulen (1984) indicate, then smokers might be better fighters than nonsmokers.

Officers and other leaders with low levels of testosterone might not behave as leaders or otherwise appear as leaders if the frequently demonstrated association of testosterone levels with social status apply to humans as well as they do to other primates (Mazur 1976). If smoking reduces testosterone levels, it is possible the leader who smokes may not achieve his potential standing in the group as a result of this diminished testosterone.

Testosterone administration has been shown to improve mood in depressed males (Itil et al. 1978, Klaiber et al. 1976). Presumably, enhanced production of this hormone associated with diminished smoking (or initiation of smoking) would be a way to maintain "good" moods which would facilitate performance of self and, in the case of leaders, also facilitate performance of subordinates.

Estradiol is a female hormone secreted by the ovaries and placenta. However, conversion of serum testosterone to serum estradiol occurs in men (and women) via aromatization (Longcope, Kato, and Horton 1969), and this process appears to be augmented by the presence of norepinephrine (Klaiber, Broverman, and Dalen 1984). They found males who smoked had significantly higher levels of serum estradiol than nonsmokers. The result held for two samples, with levels of estradiol in smokers nearly twice those of nonsmokers in both. In addition, a significant correlation was found between number of cigarettes smoked and estradiol levels in the blood. Higher norepinephrine levels in smokers (see below), may be the basis for these higher estradiol levels in smokers. One significant aspect of these higher estradiol levels in smokers relates to the very high levels of estradiol found in men with coronary artery disease (Phillips 1978, Phillips et al. 1983).

Rapid conversion of testosterone to estradiol also could account for lower serum testosterone levels in smokers.

Estrogen

Testosterone has been emphasized in this review since the bulk of soldiers are males. However, recent research (MacMahon et al. 1982) has found women who smoke have significantly lower estrogen levels than nonsmokers (estrogen is the major female hormone).[3] The differences between smokers and nonsmokers occurred during the luteal phase of the menstrual cycle (3 or 4 d after the period). Measurements made at the follicular phase (4 or 5 d before the period) did not show differences in urinary estrogen concentrations among the smoking and nonsmoking groups.

Earlier menopause of smokers has been found in numerous studies (Adena and Gallagher 1982, Daniell 1978, Kaufman et al. 1980), and this is a probable result of the diminished estrogen production in females found by MacMahon et al. Estrogen treatment of depressed females has salutary effects parallel to testosterone treatment of depressed males (Klaiber et al. 1976). Estrogen also is critical to production of strong bones both before and after the menopause (Richelson et al. 1984). In fact, a review of estrogen and mortality from all causes indicated estrogen generally facilitated good health and lower mortality rates for females who used estrogen than for those who did not (Bush et al. 1983), although a recent study by Wilson, Garrison, and Castelli (1985) found no difference in mortality from cardiovascular disease and from all causes between estrogen users and nonusers. Smoking-induced decrements in estrogen production would appear to have at least as many disadvantages for female soldiers as smoking-induced decrements of testosterone have for males (if such decrements for males occur reliably or at all).

Epinephrine (adrenaline)

Frankenhaeuser et al. (1968) found urinary epinephrine excretion increased with smoking over a no-smoking control condition and increased linearly as the number of cigarettes smoked increased from two to four to six. In a later study, Frankenhaeuser et al. (1971) again found epinephrine excretion

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[3] Given the contradictory results from "replications" of studies relating smoking to testosterone production, it probably would be important for smoking-estrogen relationships to be assessed in additional populations, as well.

increased with smoking of three cigarettes in a 50-min period compared to a no-smoking control condition. Injections of epinephrine produce cardiovascular and hand steadiness changes very similar to those produced by smoking and nicotine injection and the reliable changes in heart rate, blood pressure, and hand steadiness found with smoking appear to be mediated by the epinephrine release (Frankenhaeuser et al. 1968). Winternitz and Quillen (1977) found a small, but significant rise in epinephrine during smoking of eight high-nicotine cigarettes in 2 h. However, Carruthers (1976) failed to find significant changes in epinephrine following smoking of either three low-nicotine (.3 mg) cigarettes smoked in 30 min or three high-nicotine (1.9 mg) cigarettes smoked in 30 min, although norepinephrine levels did change.

Cryer et al. (1976) measured plasma epinephrine following smoking of two cigarettes in a 10-min period. Plasma epinephrine rose to a maximum of 113 pg per ml 10 min after smoking from 44 pg per ml before smoking. Levels remained above presmoking values for another 20 min. Heart rate and blood pressure increases preceded, but then paralleled the levels of plasma epinephrine. Sham smoking did not change plasma epinephrine levels, heart rate, or blood pressure. When the adrenergic blocking agents propranolol and phentolamine were infused prior to and during the smoking period, plasma epinephrine, heart rate, and blood pressure did not show changes with smoking. Adrenergic mechanisms were implicated in the heart rate and blood pressure increases due to the absence of such changes with adrenergic blockade. However, cardiovascular changes occurred prior to the appearance of increased epinephrine in the blood, and this indicates that it was a local excretion of norepinephrine from adrenergic axon terminals within the cardiovascular system that increased heart rate and blood pressure.

As mentioned in Chapter 6: "Effects of smoking on arousal and ability to deal with stress, pain, and fear," Vogel, Broverman, and Klaiber (1977) interpreted smoker EEG data to indicate smokers suffer from chronic underproduction of epinephrine and smoking serves to overcome this adrenergic deficiency to the subjective relief of the smoker. The immediate effect of smoking on epinephrine is an increase, as studies have shown, and this might appear to argue against the adrenergic deficiency explanation of Nesbitt's Paradox provided by Vogel, Broverman, and Klaiber. However, the serum levels of the hormone prolactin are increased immediately following smoking, yet smokers showed lower prolactin levels than nonsmokers (see references below), and it could be the same thing is true for epinephrine. The diminished capability of smokers for cerebral vasoconstriction and vasodilation found by

Rogers et al. (1984b) also may reflect an adrenergic deficiency in smokers.

Norepinephrine (noradrenaline)

Frankenhaeuser et al. (1968, 1971) found norepinephrine excretion showed no consistent trend with smoking of different numbers of cigarettes or in comparison to a no-smoking control condition. Cryer et al. (1976) did find increases in plasma norepinephrine with smoking, although they were of shorter duration than increases in plasma epinephrine. Winternitz and Quillen (1977) found no rise in norepinephrine during smoking of eight high-nicotine cigarettes in 2 h.

On the other hand, Carruthers (1976) found serum levels of norepinephrine to be significantly increased by three low-nicotine (.3 mg) cigarettes smoked in 30 min and found an even larger increase of serum norepinephrine when three high-nicotine (1.9 mg) cigarettes were smoked in 30 min. Heart rate and blood pressure changes paralleled these results for noradrenaline. Beta blockade (oxprenolol) eliminated the heart rate and blood pressure changes, but did not change the significant increases of norepinephrine as a result of smoking nor did it apparently reduce the subjective enjoyment of smoking.

Growth hormone (somatotropin)

Wilkins et al. (1982) found a 12-fold increase in growth hormone occurred 30 min after smoking two high-nicotine cigarettes (2.0 mg) and remained at elevated levels for 1 h. They found smoking two very-low-nicotine cigarettes (.2 mg) had no effect on growth hormone response. Cryer et al. (1976) also found significant increases in plasma growth hormone following smoking of two cigarettes in 10-min. Sham smoking did not increase growth hormone levels and growth hormone increases only were slightly affected by adrenergic blockade. Winternitz and Quillen (1977) found growth hormone increases beginning with the second cigarette, peaking at the fifth cigarette, and then dropping almost to presmoking levels by the eighth cigarette in the eight-cigarette, 2-h period of smoking in their study. Coiro et al. (1984) found a three-fold increase in growth-hormone 30 min following smoking of two nonfilter cigarettes in 15-min. Sandberg et al. (1973) also showed a large increase in growth hormone following rapid smoking of three cigarettes.

The implications of this large boost in production of growth hormone following smoking are not immediately clear.

Growth hormone is involved in protein and carbohydrate metabolism. Excessive growth of smokers certainly is not the result although the typical lower smoker body weight may be. For example, Janzon et al. (1983) suggested increased growth hormone release with smoking may account for the tobacco-dose-related impaired clearance of glucose from plasma that they found in their research. It is probably this impaired clearance of glucose that led to the finding of Redington (1984) of taste differences for sugar solutions between smokers and nonsmokers. Following glucose loading, smokers who smoked up to the beginning of the experimental session decreased their ratings of the pleasantness of sugar solutions. Nonsmokers and smokers who abstained from smoking prior to the session did not change their ratings of pleasantness of this solution following glucose loading. Decreased liking and consumption of sweet foods by smokers could account for their lower weight.

Cortisol

Kershbaum et al. (1968) found elevation of plasma corticosteroids 1 h following a 30-min period in which four cigarettes were smoked with an average increase of 47 percent for the nine subjects. Concentrations were lower at 2 h, but were still above initial values for six subjects. After 3 h, corticosteroid levels generally had returned to initial levels. Cryer et al. (1976) found significant increases in plasma cortisol following smoking of two cigarettes in 10-min. Sham smoking did not increase cortisol levels and cortisol increases were unaffected by adrenergic blockade. Winternitz and Quillen (1977) required subjects to smoke eight 2.5 mg-nicotine cigarettes in 2 h and found a sharp rise in cortisol between the second and third cigarette which peaked between the fifth and sixth cigarette. Wilkins et al. (1982) also found cigarette smoking (two cigarettes in 10 min) increased circulating levels of cortisol in male chronic smokers. Low-nicotine cigarettes did not increase plasma cortisol.

Seyler et al. (1984) found significant increases in cortisol with smoking of two high-nicotine cigarettes "five minutes apart." Increases in serum cortisol with smoking were particularly large when the normal or intense smoking behavior led to nausea. Adrenocorticotropic hormone (ACTH) also was found in smokers of high-nicotine cigarettes, but only when they experienced nausea. It appears the extra cortisol found in nauseous subjects was mediated by the presence of ACTH.

However, Benowitz, Kuyt, and Jacob (1984) did not find cortisol changes as a function of smoking high-nicotine cigarettes (2.5 mg) or low-nicotine cigarettes (.4 mg). Subjects smoked one cigarette every 30 min for 15 h for a total

of 30 cigarettes. Benowitz, Kuyt, and Jacob claimed earlier research found cortisol changes because of the atypical smoking of two or more cigarettes in a very brief period. Cherek et al. (1982) also found no increase in cortisol levels for a smoking period of 2 h where an average of 5.6 cigarettes were smoked compared to a control group who did not smoke.

Prolactin

Wilkins et al. (1982) found cigarette smoking more than doubled circulating levels of prolactin in male chronic smokers who smoked two 2.0 mg nicotine cigarettes within 10 min. Two low-nicotine cigarettes did not increase plasma prolactin. However, Andersen, Semczuk, and Tabor (1984) found significantly lower levels of serum prolactin in both male and female smokers compared to nonsmokers. This apparently is an instance where the acute effect of smoking is to increase hormone production, but the long-term effect of smoking is to decrease hormone production.

Vasopressin and (its carrier protein) neurophysin

Husain et al. (1975) found two nonfilter cigarettes smoked in 15-min produced significant boosts in both serum vasopressin and serum neurophysin with peak concentrations occurring in 5 to 15 min after the second cigarette. A rapid drop in both substances then occurred. The high degree of correlation of the two substances during this smoking-related increase and subsequent decrease following smoking indicated the close linking of their release mechanisms. Prior administration of ethanol eliminated or blunted vasopressin and neurophysin increases.

Rowe, Kilgore, and Robertson (1980) found smoking of one "high"-nicotine cigarette (1.2 mg) and smoking of one low-nicotine cigarette (.6 mg) produced reliable increases in plasma vasopressin. However, intravenous infusion of a solution containing 2 mg of nicotine did not produce a significant increase in plasma vasopressin. The failure of intravenous nicotine to mimic smoking effects on vasopressin led these researchers to conclude that an airway-specific mechanism was the basis for the nicotine effect. However, blood pressure increments with intravenous nicotine were smaller than for smoking of even the low-nicotine cigarette and it is probable the bolus of nicotine to the brain is larger following smoking than following intravenous injection of 2 mg. of nicotine over a 5-min period.

Pomerleau et al. (1983) measured vasopressin and neurophysin and found both were stimulated by smoking moderate-nicotine (1.46 mg) and high-nicotine (2.87 mg) cigarettes. Nicotine levels in the blood also were measured and were correlated significantly with blood levels of vasopressin and neurophysin. As these authors pointed out, vasopressin release has been associated with improved cognitive functioning. It also appears to mediate the changes in skin blood flow associated with smoking (Waeber et al. 1984).

Beta endorphins

Evidence is accumulating that beta-endorphins are released during smoking. These are the brains natural opiates and have been implicated in such things as pain tolerance (Pomerleau, Turk, and Fertig 1984). Karras and Kane (1980) inferred smoking increased beta-endorphins when they found an endorphin "blocker" (naloxone) reduced smoking and the desire to smoke. Malizia et al. (1978) reported acupuncture-elicited-analgesia, which causes a revulsion for tobacco in at least some tobacco addicts, lost this effect following naloxone administration and, in fact, acupuncture actually increased the desire to smoke. This, too, was hypothesized to indicate that smoking increases endorphin production.

Tobin, Jenouri, and Sackner (1982) found naloxone blocked the depression of respiration that normally occurs with smoking (and which may be a factor in the perceived reduction in emotionality with smoking). They concluded that the normal smoking-related depression of respiratory drive is one result of endorphin production.

Pomerleau et al. actually measured beta-endorphins and found these endogenous peptides, but not ACTH, were stimulated by smoking moderate-nicotine (1.46 mg) and high-nicotine (2.87 mg) cigarettes. Nicotine levels in the blood also were measured and were correlated significantly with serum beta-endorphin levels. These studies that show the production of beta-endorphins by smoking and the probable role of these in addictive smoking suggest jogging, acupuncture, and other nonsmoking means for producing beta-endorphin release should assist in smoking-cessation programs (Chernick 1983).

Other hormonal changes

Gofin et al. (1982) reported higher serum thyroxine levels in female smokers and Melander et al. (1981) reported abstention from smoking was accompanied by small reductions in thyroxine and rT_3 and concluded that smoking "promotes a

modestly increased secretion of thyroid hormone ..." Christensen et al. (1984) found female smokers to have higher serum T_3 and lower rT_3, no change in thyroxine, and a higher incidence of goiters than nonsmokers. Sepkovic, Haley, and Wynder (1984) found heavy smokers had significantly lower levels of thyroxine and significantly lower levels of another thyroid hormone, T_3--triiodothyronine, than nonsmokers. These differences were on the order of ten percent and it was argued that even these small changes may have large ramifications for overall metabolism as well as influencing both androgenic and estrogenic steroid activity. Cyanide compounds in cigarette smoke, which may inhibit the recycling of thyroidal iodine, were seen by Sepkovic, Haley, and Wynder as possible determinants of reduced thyroid hormone levels with smoking. Much additional research needs to be done to explain or resolve contradictory results related to smoking and thyroid function.

Conclusions and military implications

The well-established increases in epinephrine (adrenaline) production immediately following smoking and the adrenergic deficiency following long-term smoking proposed by Vogel, Broverman, and Klaiber (1977), could go a long way toward explaining smoking withdrawal symptoms, reduced blood pressure and heart rate with smoking deprivation, decreased cerebral vascular capacity in smokers, and other puzzling aspects of smoking. The surprising fact that habitual smokers do not wake up every 30 min for a cigarette might be related to a reduced requirement for cerebral circulation during sleep in both smokers and nonsmokers. During waking hours, habitual smokers help meet cerebral circulation requirements by smoking with its associated boost of epinephrine. Long-term smoking reduces the capacity for "nonboosted" vasodilation of the cerebral vascular system (Rogers et al. 1984a, 1984b, Wennmalm 1982), and it is probably this blood-deprived brain of the deprived smoker that produces many of the unpleasant smoking-withdrawal symptoms. Fortunately, quitting smoking restores cerebral circulation to levels approaching those of nonsmokers (Rogers et al. 1985).

The contradictory results related to the effects of smoking on testosterone levels and for levels of thyroid hormones apply to a lesser extent for most other hormones. Despite a large number of studies in this area, relatively little is known about smoking and its effects on hormone production. And also little is known about the effects of these hormonal changes, particularly over the long term. Continuing development of more refined and economical techniques for measuring hormones should accelerate research in this area.

The sex hormone diminution associated with long-term smoking that occurs for women (MacMahon et al. 1982) would appear to have information value in programs designed to reduce smoking. The five to ten less yr of life which, on the average, can be expected for the heavy smoker (Rogot 1978), may mean less to an 18-yr-old girl (who sees even a premature death as far in the future) than the prospect of diminished femininity associated with a smoking-related reduction of major female sex hormone levels.

However, such a ploy might be dishonest for male smokers. More and more recent evidence shows higher testosterone levels in male smokers than male nonsmokers with one study showing the pattern in normal men at all ages (Deslypere and Vermeulen 1984). Several review and popular articles (e.g., Willenbecher 1979), based largely on one or two of the early studies that claimed lower testosterone production in smokers, recommended men quit smoking if they wanted to preserve their masculinity. One wonders whether the desire for effective antismoking propaganda may not have somehow influenced the results of studies showing lower testosterone in smokers compared to nonsmokers. Research is needed to clear up these contradictions related to long-term and short-term effects of smoking on testosterone production (see Chapter 12: "Needs for additional research on smoking and soldier performance").

Chapter 8

The effects of tobacco deprivation

Tobacco deprivation for the habitual smoker typically leads to a highly stressful experience involving alterations of mood and concentration (Myrsten, Elgerot, and Edgren 1977). Performance of some tasks has been shown to deteriorate during smoking abstinence (Heimstra et al. 1980, Wesnes and Warburton 1978). Some studies have shown tolerance of pain and tolerance of other stressors are reduced following brief periods of smoking abstinence (Nesbitt 1973). Many smokers would like to quit smoking, but find the effects of withdrawal too unpleasant to bear, and failure rates in smoking cessation programs typically are greater than 80 percent (Lichtenstein 1982).

However, Pertschuk et al. (1979) studied the incidence of alcohol use, psychotropic medication, and use of mental health facilities, among people who participated in a smoking cessation program. He found no evidence of increased use of the above and concluded withdrawal effects are not as stressful as popularly depicted. However, people forced to abstain from smoking, such as soldiers in combat or wearing protective clothing, might not have as much acceptance or tolerance of unpleasant withdrawal effects as these people who were attempting to reduce smoking.

Military situations that prevent smoking

The actual consequences of tobacco deprivation (which West [1984] concluded is primarily nicotine deprivation) for civilian activities and for most peacetime military activities probably are not great, simply because there is little tobacco deprivation, at least, for people who can afford tobacco. Nonsmoking work situations are increasing, but smokers typically can find opportunities to take breaks in smoking areas.

Civilian outdoor activities typically provide fewer instances of smoking deprivation. However, smoking is highly dangerous in outdoor tactical military settings. The flame of matches or lighters and the glow of burning tobacco are highly visible both to the eye and to devices which amplify light and infrared energy. As described earlier, the saying, "Three on a match is bad luck," indicates soldiers sometimes have smoked openly in combat despite the danger. A craving strong enough to lead to dangerous smoking probably has other detrimental effects on the performance of the soldier who is deprived of

tobacco. At a minimum, it might distract him from watching for the enemy.

Another military situation that precludes smoking is wearing of the protective mask (gas mask) and other protective clothing. Smokers probably experience much more discomfort and diminished performance than nonsmokers while wearing protective clothing for extended periods due to tobacco withdrawal effects and, for long-term smokers, possibly also due to smoking-diminished lung capacity (see Chapter 12: "Needs for additional research on smoking and soldier performance").

Effects of deprivation on physiological processes and subjective symptoms

Halama (1980) predicted large performance degradation would occur for soldiers who smoked in situations where smoking was impossible. However, very little research on the effects of tobacco deprivation on military task performance was found, either to support or counter this prediction. In a study of submarine personnel who were prevented from smoking for 72 h, Weybrew and Stark (1967) found deprived smokers rated themselves as more tired and less friendly than nonsmokers during the deprivation period. Relative to nonsmokers, deprived smokers also ate more, reported poorer sleep, reported poorer moods, found it more difficult to concentrate, and reported more "nauseous symptoms" during the deprivation period. Reductions in pulse rate for deprived smokers during the deprivation period also were found. Critical flicker frequency did not differ between deprivation and smoking conditions for the smokers. Measures of task performance were not included in this study. No other studies of smoking deprivation effects on military personnel were found and the remainder of this chapter will discuss research on tobacco deprivation in laboratory and other nonmilitary settings which may have implications for military operations.

Gilbert and Pope (1982) studied the effects of 24 h of smoking withdrawal on physiological responses of smokers. Unlike many studies of deprivation effects which deal with people in smoking cessation clinics, these men had no intention of quitting smoking except for the day of the study. Gilbert and Pope found changes in these physiological responses on the 1 d of smoking cessation were marked and generally indicated lower activation of the physiological system. Heart rate decreased, finger temperature increased (indicating improved peripheral circulation), and postural tremor decreased, "... meaning that hand-eye coordination and other kinds of dexterity may be facilitated." On the negative side was the unpleasant craving for cigarettes which increased throughout the depriva-

tion period. Unfortunately, no performance tasks were included to measure possible changes in performance as a function of withdrawal.

Shiffman and Jarvik (1976) studied 35 volunteers in a smoking cessation clinic over an abstinence period of 12 d. Craving for cigarettes, alertness (stimulation), psychological symptoms, and physical symptoms were measured by questionnaire. Only craving scores changed over the period. Sharp drops occurred for the 11 subjects who were totally abstinent over the period with craving at low levels by Day 8. For the partially abstinent group (24 subjects), craving levels remained high for the total period. The difference between the partially and totally abstinent groups was described as indicating that craving drops rapidly when abstinence is total. However, a more probable interpretation of the results is abstinence is much more difficult when craving is high. Support for this latter explanation derives from the fact that differences already appeared in craving between totally and partially abstinent groups on the first d following abstinence on which craving ratings were made.

A large number of measures were made over 4 d of tobacco deprivation by Hatsukami et al. (1984), who randomly assigned subjects to deprivation and control groups and who included nearly every variable reported to change in previous research on tobacco deprivation. Subjects showed significant changes on only a small number of these variables following smoking deprivation that were not paralleled by similar changes in the smoking control group that also was confined for 4 d. Among the significant changes, heart rate dropped from an average of 80.7 bpm for the smoking baseline to an average of 70.0 bpm for the 4 d of abstinence. The major drop in heart rate occurred on the first d of withdrawal. Heart rate continued to drop for the next 2 d then showed a small rise on the fourth d. Craving for cigarettes, confusion, and "depression-dejection" all increased significantly compared to control subjects who smoked during the withdrawal period. Craving, confusion, and depression-dejection reached maximum change on the second d of withdrawal with small "rebounds" over the next 2 d.

Of particular relevance for soldier performance was a significant increase in reported number and duration of awakenings during sleep during the deprivation period found by Hatsukami et al. Both of these variables reached their peak on the second d of abstinence. Unfortunately, no objective data on sleep patterns were obtained in the study.

A study by Hughes et al. (1984b) from the same laboratory as Hatsukami et al. confirmed many of the Hatsukami et al. findings following smoking withdrawal. Hughes et al. also

showed amelioration of many smoking-withdrawal symptoms with administration of nicotine chewing gum. This indicated the key role of nicotine deprivation in smoking withdrawal symptoms. However, significant reductions in heart rate and hand tremor still occurred for subjects using nicotine gum (and placebo gum) during smoking abstinence, indicating nicotine from gum does not match smoking-generated nicotine in its effects on some physiological functions.

Schneider and Jarvik (1984) also found unpleasant subjective symptoms associated with smoking withdrawal generally were reduced for a group receiving nicotine gum compared to a group receiving placebo gum. Withdrawal symptom ratings rose sharply over symptom ratings reported during baseline smoking for both the nicotine and placebo groups. Levels on the first two measures on Day 1 actually were higher for the group chewing nicotine gum. Following this, however, the nicotine gum group reported less discomfort than the placebo gum group over 5 d of abstinence. Withdrawal symptoms were found to be higher in the evening than in the afternoon, particularly for the placebo group.

Myrsten, Elgerot, and Edgren (1977) found urinary epinephrine levels to remain below smoking levels for 5 d of abstinence. West et al. (1984c) studied urinary epinephrine levels over a longer period of smoking abstinence and found epinephrine dropped during the first 3 d of abstinence of smoking among a group of heavy smokers. Abstinence continued for at least 10 d, and urinary epinephrine levels in a significant number of subjects showed a rebound effect when measured on Day 10. The strong influence of epinephrine in many of the cardiovascular changes with smoking suggests these too would show a rebound effect. West et al. collected data on heart rate, skin temperature and urinary cortisol concentrations and reported "in some cases these variables did appear to follow a time course which might suggest a rebound, ..." but there was no unequivocal statistical support for this. However, the rebound on Day 4 of deprivation found by Hatsukami et al. for heart rate would support this suggestion of a rebound for heart rate as well as epinephrine in the West et al. research. As West et al. point out, these rebound effects suggest the initial drops in epinephrine during smoking abstinence found in this and other studies (e.g., Shiffman 1979) reflect an abnormal state for the habitual smoker resulting from withdrawal from smoking, and not just a return to normal nonsmoker endocrine levels and physiological states.

One study looked at the effects of 30 d of deprivation on cardiovascular and other variables. Glauser et al. (1970) found heart rate to be significantly lower following this long

abstinence period. This indicates any rebound effect on heart rate does not return heart rate to levels prior to smoking.

In the most recent study found on deprivation effects, Cummings et al. (1985) looked at specific smoking withdrawal symptoms over a 21-d period of abstinence. Irritability was the symptom most frequently reported followed by feeling sleepy, coughing, sleeplessness, constipation, tightness in the chest, dizziness, and mouth sores. Some of these such as coughing, constipation, and mouth sores are questionable withdrawal symptoms, but coughing did decline linearly over the 21-d period. They found subjects reported high levels of irritability which also declined linearly over the 21-d period. The same was true for "feeling sleepy," dizziness, and tightness in the chest. The average number of symptoms declined linearly over the 21-d period, as did reported mean craving for a cigarette, but there was a trend for craving to increase during the second week, then resume its decline. At the end of the 21-d period, few symptoms were experienced and craving was experienced only occasionally. There was a trend to more symptoms and craving for heavy than light smokers.

Effects of tobacco deprivation on performance

Heimstra, Bancroft, and DeKock (1967) compared groups of smokers, deprived smokers, and nonsmokers and found the deprived smokers to be inferior to the other two groups on the tracking task and a reaction-time task included in the test battery of their study. However, these differences between deprived and nondeprived smokers were found during the first h of testing and it may be that these deprived smoker differences from nonsmokers at least partly reflect different initial levels of performance of the different groups. Subjective mood changes also were measured by Heimstra, Bancroft, and DeKock and found to be greatest for the deprived smokers, with significant increases in aggression and fatigue and significant decreases for concentration and social affection. Aggressiveness also increased sharply with deprivation in the research of Schechter and Rand (1974). These mood/concentration/aggressiveness changes with abstention from smoking may be the key to the performance degradation shown by deprived smokers.

In a later study, Heimstra et al. (1980) found deprived smokers showed significant decrements in performance on a perceptual-motor tracking task compared to the performance of nondeprived smokers. The task required tracking of the changes in orientation of a target needle (which rotated like a hand on a clock) by covering it with another "cursor" needle. The cursor needle was connected to a hand-controlled tracking knob. Occasional mental addition, vigilance, and reaction time

tasks added to the difficulty of the tracking task. For male deprived smokers, time on target was more than 20 percent less than for nondeprived male smokers at the end of the 3-h session although nearly half of this difference already was found during the first 30 min after smoking a pretest cigarette, suggesting withdrawal effects influenced performance by only about 10 percent. For female deprived smokers, significant performance degradation of about 10 percent was found in mid to later stages of the tracking task. Unlike for the males, female deprived smokers did not differ from smokers during the first 30 min. Performance on the concurrent tasks frequently showed larger decrements for deprived smokers at the later stages of the 3-h testing session, but, unlike performance on the tracking task, the differences were not significant. The authors concluded, "under restriction (of smoking), workers who normally smoke may experience lowered arousal, increased stress, decreased job satisfaction and a number of other undesirable effects. These behavioural outcomes should certainly be avoided as they may lead to more critical events. These effects may well be manifested by decreased productivity and increased accidents."

Mertens, McKenzie, and Higgins (1983) compared 17 habitual smokers on the performance of aviator-related tasks with and without smoking during 30-min breaks during the 4-h test session in a similar paradigm to that of Heimstra et al. (1980) except subjects served as their own controls in separate counterbalanced smoking and smoking-deprivation conditions. A tracking task showed significant decrements in the no-smoking condition and overall performance on several concurrent tasks (including the tracking task) also was significantly worse without smoking during breaks. The trend for the complex of tasks was for subjects while not smoking to become progressively worse over time on the tasks relative to their smoking performance.

Mertens, McKenzie, and Higgins found workload changes (changes in the number of different tasks being performed) produced error scores for tracking performance that ranged from 338 for low workload to 747 for high workload. These dwarfed smoking deprivation effects of about six percent on tracking (error score of 557 for smoking compared to 592 for no-smoking). Even time on the tracking task produced twice as large an effect as the smoking manipulation (error score of 535 for Period 1 versus 614 for Period 6). Contrary to the conclusions of Mertens, McKenzie, and Higgins, the statistically significant decrement with nonsmoking appears to be small from a practical viewpoint. What is more, for the key tracking task, a five percent difference in favor of nondeprived smokers appeared even during the first 30 min after the smoking that preceded the 4-h deprivation period. This almost suggests

subjects were consciously or unconsciously influenced in their performance by knowledge of the condition in which they were participating.

As mentioned in a previous section, smokers occasionally have outperformed nonsmokers on certain perceptual and speeded response tasks (Wesnes and Warburton 1978). However, differences between smokers and nonsmokers that favor smokers generally are small. However, on almost every laboratory task of more than a few min duration, deprived smokers have performed worse than nondeprived smokers. The increasing confrontations between smokers and nonsmokers support the notion smokers are members of a fraternity of sorts. At least some smokers are apt to be more concerned about their performance when they are in the smoking group (of between-group comparisons of nondeprived smokers and deprived smokers) or when they are in the smoking condition (in repeated-measures paradigms for exploring these differences). It is less probable smokers would be biased to perform better in the condition where they are not allowed to smoke. Just a small percentage of smokers in an experiment, who consciously or unconsciously worked harder or faster when smoking or who consciously or unconsciously worked less hard or more slowly when not smoking, could provide the pattern of results shown by Heimstra, Bancroft, and DeKock (1967), Heimstra et al. (1980), and Mertens, McKenzie, and Higgins (1983) where deprived smokers' performance is inferior from the outset of long tasks.

However, even people who are for intoxication probably cannot bias results enough to overcome intoxication effects and if smoking were to be detrimental to performance to any substantial degree, even nondeprived smokers would show the effect compared to their deprived performance. Elgerot (1976) found nondeprived smokers performed significantly worse than deprived smokers on Raven's Progressive Matrices, which is a difficult problem-solving task used to measure intelligence. Elgerot found abstinence from smoking also facilitated performance on two other reasoning tests for these subjects compared to the smoking condition. Simpler tasks such as marking each "n" and "e" in meaningless rows of letters or proof-reading were facilitated slightly by smoking, although these advantages for nondeprived smokers were not significant.

Elgerot interpreted her results in terms of optimal levels of arousal and the "arousing" effects of smoking. For complex intellectual tasks, she suggested smoking subjects were too aroused for optimal performance. Unfortunately, no physiological or subjective arousal indices were included in her research to confirm this hypothesis that nondeprived smokers were highly aroused. It could be the other way around. Reduced arousal for the smokers may have left them with too little arousal to

optimally perform on the complex intellectual tasks whereas arousal associated with smoking deprivation may have boosted performance. Kleinman, Vaughn, and Christ (1973), for example, claimed deprived smokers were too aroused for a difficult paired-associate learning task, but optimally aroused for a simple one.[1]

Although this section has primarily described decrements and potential decrements in performance as a result of smoking deprivation, some short-term effects of smoking withdrawal would be expected to highly benefit performance of soldiers in combat. Improved physical performance with only 1 d of smoking deprivation was found by Rode and Shephard (1971). This research has been described more fully in Chapter 2: "Effects of smoking on physical work capacity and endurance." Davies et al. (1979) found significant increases in the availability of oxygen following 48 h of withdrawal from smoking that averaged eight percent. Such increased availability of oxygen was seen as improving the chances of survival in surgical operations and this has direct implications for surviving battlefield injuries, as well. The probable improved marksmanship and reduced chance of frostbite following deprivation are described in the section on military implications.

Will deprivation reduce effectiveness of pilots who smoke?

Several years ago, it was proposed airline pilots not only refrain from smoking while flying, but abstain for at least 8 h prior to flying (see Dille and Linder 1981). Evidence in support of this proposal (and against smoking) was provided in a review by Robinson and Wolfe (1976). Dille and Linder provided a subsequent review of smoking and aircraft accidents and of literature on smoking effects that led to the conclusion that smoking withdrawal effects in the pilots and other personnel forced to abstain probably would cause a greater problem for aviation safety than the small changes in performance during or following smoking. Both reports may have reflected biases of the authors, with one against and one for smoking. Robinson and Wolfe did not include many of the research articles in their review that have either shown no effect of smoking on performance or have shown an enhancement of performance following smoking (see Chapter 3: "Effects of smoking on perceptual processes"). Dille and Linder, however, tended to overemphasize the negative results Robinson and Wolfe

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[1] A problem exists with this explanation since high arousal "helps" for the difficult Raven's task of Elgerot (1976) and "hurts" for the difficult paired-associates task of Kleinman, Vaughn, and Christ (1973).

(1976) missed, and failed to note the fact that pilots would be "backed up by one or two additional crewmembers" would protect against withdrawal effects on performance as well as deleterious effects of smoking.

Smoking deprivation may influence ocular accommodation and conversely, there may be immediate effects of smoking on accommodation (see Chapter 3: "Effects of smoking on perceptual processes"). Test pilot Chuck Yeager repeatedly has claimed his ability to control the focus of his eyes and focus at infinity was a major factor in his ability to detect enemy aircraft at great distances and his resultant success as a fighter pilot in World War II and Korea. This same ability would allow additional time for pilots to detect other aircraft and avoid midair crashes. Until research is conducted that identifies the probable critical role of smoking and smoking deprivation on ocular accommodation, the question: "Will deprivation reduce effectiveness of pilots who smoke?" cannot be answered.

Conclusions and military implications

Anecdotal evidence indicates craving for cigarettes in some individuals deprived of smoking is so intense it can be used to extract information. In a program designed to train soldiers to withhold information from the enemy, at least one "captured" soldier, who was deprived of tobacco for a few h by his captors, actually compromised unit security in return for tobacco (Oberholtzer, personal communication 1983). One military implication of nicotine gum and other nonsmoking forms of nicotine such as aerosols (West et al. 1984a) is these smoking-substitutes can be used to reduce craving in smoking soldiers when they are in situations where they cannot smoke.

The large increase in cigarette craving and in other unpleasant symptoms following smoking deprivation in habitual smokers also will take some toll on performance, particularly on boring or arduous tasks that produce their own share of unpleasant symptoms. However, the decrements in performance with smoking deprivation in laboratory tracking tasks are typically only about 10 percent, and usually are outweighed by performance decrements over time or by the effects of adding concurrent operator tasks. These small changes may not be sufficient to justify smoking when other factors argue against it.

One factor that may justify the imposition of smoking abstinence in combat settings is the reduction of postural tremor that occurs with smoking abstinence of a few h. This could improve significantly rifle shooting and firing of other

hand-held weapons, although research is needed to examine these probable effects of smoking and smoking deprivation on soldier marksmanship. Still another definite benefit associated with smoking deprivation will be improved performance on extended physical tasks (if smokers don't react negatively to abstinence) and the longer the deprivation, the greater the improvement. Improved peripheral circulation in deprived smokers argues for curtailing or prohibiting smoking during cold-weather operations to reduce the danger of frostbite. As was described in Chapter 3: "Effects of smoking on perceptual processes," abstinence also may increase visual sensitivity of smokers.

Another possible factor arguing for smoking abstinence is truly difficult problem-solving tasks such as Raven's Progressive Matrices may be performed better following a period of smoking abstinence. If abstinence enhances performance on difficult problem-solving tasks, Army leaders and operators of complex Army weapons systems (e.g., Patriot) should refrain from smoking during key engagements. However, more research is needed on smoking and smoking abstinence effects when tasks are difficult since some contradictions exist among results of the few available studies. Actual leader decision-making tasks and actual complex operator tasks used in Army training would increase the validity of such research results.

Chapter 9

Smoking-disease relationships: Effects on productivity and absenteeism

The 40-yr-old who smokes two packs of cigarettes daily can expect to live 8.8 yr less than the nonsmoking 40-yr-old (Rogot 1978). Death from lung cancer is more than ten times as likely to occur in smokers than in nonsmokers (Rogot and Murray 1980). They also report mortality from heart disease and emphysema is much higher in smokers than nonsmokers. However, most of the deaths and debilitation produced by lung cancer and emphysema occur after soldiers and officers have left the active Army. The impact of these two smoking-related-diseases on medical costs for retired military personnel is immense. On the other hand, Leu and Schaub (1983) analyzed medical costs and concluded lifetime medical costs are higher for nonsmokers than for smokers because smokers' higher annual utilization rates are overcompensated for by nonsmokers' higher life expectancy. Smokers, by dying earlier, also won't collect military retirement as long as nonsmokers. However, cost matters are beyond the scope of this report.

This chapter will describe smoker-nonsmoker differences in diseases other than cancer and emphysema.[1] Special emphasis will be placed on research data collected from military populations and on diseases or other medical conditions, such as frostbite, which are particularly salient for military performance. The section on disease will be followed by a review of smoking-related absenteeism due to illness (and other factors) as it has been measured in military and civilian settings.

Increased incidence of disease among smokers

Heart disease: As with lung cancer and emphysema, most victims of heart disease have retired from the military before this disease takes its toll. However, coronary artery disease is by far the major cause of death (Rogot and Murray 1980), and a substantial number of young and middle-aged men do incur fatal and nonfatal heart attacks. Heart disease probably is the most important cause of death and serious disability among military personnel in times other than war and, the younger the victim, the greater the likelihood smoking was a factor. This was most

[1] Fielding (1985a) has provided a recent brief review that describes the strong relationship of smoking to lung cancer, to other cancers, and to obstructive lung disease. A more extensive review is available from Clee and Clark (1982).

clearly shown by Kaufman et al. (1983) who studied the incidence of myocardial infarction in men between the ages of 30 and 54 and related it to cigarette smoking and to the nicotine, tar, and carbon monoxide levels of the cigarettes smoked. Among 30-44 yr olds, smokers of 35 or more cigarettes daily were more than seven times as likely to have a myocardial infarction than men who had never smoked. Even smokers who smoked less than 25 cigarettes daily were 4.6 times as likely than nonsmokers to have a myocardial infarction. In the 45-54 age category, nonsmokers had a somewhat higher incidence of myocardial infarction relative to smokers, but smokers of more than 35 cigarettes daily still were more than 2.5 times as likely to have a myocardial infarction than men who had never smoked.

Kaufman et al. also studied exsmokers and found they had an incidence of myocardial infarction only slightly higher than nonsmokers in the 30-44 age group and nearly identical to nonsmokers in the 45-54 age group, indicating the importance of quitting smoking for prevention of myocardial infarction in young and middle-aged men. They did not find differences in tar, nicotine, and carbon monoxide content of cigarettes were related to the incidence of myocardial infarction, apparently indicating the futility of brand-switching as a health measure for coronary artery disease prevention.

Bush and Comstock (1983) found similar significant associations between smoking and cardiovascular disease for women 25-44 and 45-64 yr. For all arteriosclerotic heart disease deaths, the relative risks associated with smoking more than 20 cigarettes daily were 3.6 and 2.2 for the above two age groups. For sudden deaths from arteriosclerotic heart disease, the relative risks were 6.5 and 2.7. Women older than 64 did not show a relationship of smoking to heart disease mortality.

A recent study by Rosenberg et al. (1985) showed dramatic reductions in the risk of myocardial function occurred when smokers abstained from smoking. For men who had smoked in the previous yr, the estimated relative risk of myocardial infarction was 2.9. Among exsmokers who had abstained for 12 to 23 mo the estimate was 2.0, and for those who had abstained for longer intervals the estimates were about 1.0, indicating a level of myocardial function similar to that in men who had never smoked.

A high concentration of high-density-lipoprotein cholesterol (HDL-C) in the blood is associated with a decreased probability of heart disease (Stamford et al. 1984b). Smoking has been associated with low concentrations of HDL-C and Stamford et al. (1984a, 1984b) found that although exercise and alcohol consumption were associated with higher levels of

HDL-C, men and women who exercised, consumed alcohol, and smoked still averaged lower levels of HDL-C than men and women who exercised, consumed alcohol, and did not smoke.

Before leaving heart disease, some intriguing results of Friedman et al. (1975, 1983) deserve mention. These authors found smokers were nearly twice as likely to have myocardial infarctions as nonsmokers. They also found smokers were more likely to worry about many aspects of their lives (money, business, etc.) than nonsmokers. Their particularly interesting finding, however, was that those relatively few smokers who were not worriers were considerably more likely to have heart attacks than smokers who did a normal amount of worrying. On the other hand, nonsmokers when they had heart attacks were more likely to be worriers than were nonsmokers without myocardial infarctions. Friedman et al. (1983) discuss the need for more research on this interactive relationship between smoking, worrying, and heart disease.

Stroke: Strokes are the most common cause of chronic disability in the Western world (MacKay and Nias 1979). These authors also reported one-fourth to one-third of all strokes occur in men and women under 65, and about six percent occur prior to age 50. Given these figures, some military personnel are undoubtedly victims while still on active duty. MacKay and Nias reported 70 percent of stroke victims aged 65 and under were smokers compared to 41 percent of a control population. They also found 48 percent of stroke victims were heavy smokers (more than 20 cigarettes daily) compared to only 18 percent of the controls.

Bell and Ambrose (1982) found smoking increased the risk of sustaining cerebral infarction by a factor of 1.9 for men and 2.4 for women. Smoking was not found to be a factor in primary intracerebral hemorrhage unlike subarachnoid hemorrhage where smokers carry a relative risk approaching four times that of nonsmokers. In another study, Salonen et al. (1982) found smoking increased men's risk of sustaining cerebral infarction by a factor of 4.2 and the risk of other strokes by 2.2. Smoking was not a risk factor for strokes in women.

Reduced cerebral blood flow in smokers relative to nonsmokers (Rogers et al. 1984a) and reduced capacity for increasing and decreasing cerebral blood flow following breathing of CO_2 and oxygen, respectively (Rogers et al. 1984b), are undoubtedly factors in this increased risk of stroke for smokers. Rogers et al. (1985) have shown abstention from cigarette smoking even after three or four decades of smoking significantly increased cerebral blood flow, although not to the levels of subjects without a history of cigarette

smoking. Presumably, risk of stroke decreased as well in these people who quit smoking.

Respiratory disease: A number of studies, described by Finklea et al. (1971), have shown smokers are more prone to respiratory infections than nonsmokers while other studies have shown no association. They studied acute upper and lower respiratory infections in cadets at The Citadel and found the incidence rates for both to be significantly higher for smokers. Smokers were 1.29 times as likely to contract upper respiratory illness than nonsmokers. For the lower respiratory tract, smokers were more than twice as likely to suffer illness as nonsmokers. They attribute their positive results partly to the homogeneity of their population which reduced potentially important sources of variation such as age, sex, and socioeconomic background which may have overwhelmed smoking effects in other studies that have failed to show a smoking effect on respiratory disease. They also noted that this difference appeared despite the rigorous physical conditioning of both smoking and nonsmoking cadets.

Aronson et al. (1982) found women with acute respiratory tract illness were nearly twice as likely to smoke as women in a control group (57 percent versus 34 percent). Of the 867 men and women with acute respiratory tract illness included in the study, 58 percent were smokers and these smokers had a significantly longer duration of cough and a significantly greater frequency of abnormal auscultatory finding than did the nonsmokers. Smith et al. (1981) made similar observations on 1,867 staff members of 12 Australian industries. Respiratory symptoms were significantly higher for smokers than nonsmokers both in men and women. Smoking was related particularly to cough frequency and sputum production. Chronic respiratory diseases, such as chronic bronchitis, also are much more frequent among smokers than nonsmokers and exsmokers (Jedrychowski 1976, McClimans et al. 1984).

Low back pain: Frymoyer et al. (1983) found a strong association between smoking and the incidence and severity of low-back pain. They found 39.6 percent of men without back pain were smokers, 43.8 percent of men with moderate back pain were smokers, and 53 percent of men with severe back pain were smokers. These results confirmed an earlier study showing smoking to be associated with back pain (Frymoyer et al. 1980). Frymoyer et al. (1980) speculated smoking may reduce blood flow to the vertebrae rendering the disc more susceptible to mechanical deformities. Such an explanation also may apply to results of a study by Kelsey et al. (1984) who reported smoking was associated with the incidence of acute prolapsed lumbar intervertebral discs and the more cigarettes smoked, the higher the incidence. These authors did not provide any

definite explanation of their results, but increased coughing by smokers was suspected even though reported coughing showed no association with disc prolapse in their study.

Headache: Schele, Ahlborg, and Ekbom (1978) found 50 percent of headache suffers smoked compared to 39 percent of controls. Markush et al. (1975) found women who smoked were significantly more apt than nonsmokers to report two or more migraine headache symptoms (27 percent versus 19 percent). Moilanen et al. (1976) studied young Finnish soldiers and found 43 percent of smokers and 27 percent of nonsmokers reported frequent headaches. Other research results reported by Schele, Ahlborg, and Ekbom indicated no association with smoking (Ogden 1952) or a smaller incidence of migraine headaches among smokers than nonsmokers (Volans and Castleden 1976).

Frostbite: The sharply reduced peripheral blood flow associated with smoking (e.g., Waeber et al. 1984) and the related slow recovery in finger temperature after cold exposure following smoking (Cleophas, Fennis, and van't Laar 1982) both would be expected to increase susceptibility of smokers to frostbite. Sumner, Criblez, and Doolittle (1974) found an association between smoking and the frequency of frostbite in a soldier population in Fort Wainwright, Alaska. Smokers had a higher incidence of frostbite than nonsmokers. White soldiers showed this effect more strongly than blacks. The effect was found both for soldiers above and below age 25. The effect was strongest among light smokers (less than one pack daily). Heavy smokers (one pack or more daily) had a lower rate of frostbite than the light smokers. Miller and Bjornson (1962), on the other hand, found it was the heavy smokers who differed from nonsmokers (and light smokers) and they suggested the higher rate of cold injury was related to heavy smoker's greater tendency to smoke in freezing situations and to the acute reduction of peripheral blood flow by smoking.

Schuman (1953), as reported by Sumner, Criblez, and Doolittle (1974), did not find a relationship of smoking to incidence of frostbite among US soldiers in Korea. Loesser (1944), in a study described by Miller and Bjornson (1962), did not find World War II German soldiers who smoked to have any higher incidence of cold injury than nonsmokers. However, no study has shown smoking to reduce cold injury and given the large decreases in circulation to the extremities with smoking, it is probable that smoking is highly dangerous during environmental conditions than can lead to cold injury. More data are needed to resolve the questions raised by the apparent contradictions in these research results. Different areas of the body which receive frostbite injury should be considered in this needed research, given the results of Suter, Buzzi, and Battig (1983), who found vasoconstrictive responses to nicotine

were "considerable with the finger recordings, modest with the foot recordings, and absent with the forehead and the ear recordings."

Peptic ulcer: An association between smoking and peptic ulcers has been established in a number of studies (US Department of Health, Education, and Welfare 1979). Sandberg and Bliding (1976) found recruits and NCOs in Swedish training battalions who smoked heavily (more than 15 cigarettes daily) were more apt to have ulcers than nonsmokers. Kikendall, Evaul, and Johnson (1984) have provided an extensive review of smoking and gastrointestinal physiology and nonneoplastic digestive disease that primarily deals with peptic ulcer disease. McCarthy (1984) also summarized research on smoking and ulcers and found smokers were more prone to peptic ulcers, the amount of smoking was associated with ulcer frequency, and smoking impaired spontaneous and drug-induced ulcer healing. However, Barakat, Menon, and Badawi (1984) found no significant difference in healing rate for ulcer patients who were smokers and ulcer patients who were nonsmokers. However, they did not look at smoking abstinence effects on ulcer healing.

Influenza: Kark and Lebiush (1981) found an outbreak of influenza-like disease among an Israeli training unit of 176 female recruits was much more apt to strike smokers than nonsmokers (60.0 percent versus 41.6 percent). The disease also was more severe for smokers than nonsmokers with 83.3 percent of the smokers visiting the clinic having the disease compared to 59.6 percent of nonsmokers.

Acquired immune deficiency syndrome (AIDS): Newell et al. (1985) found 52 percent of male homosexuals with AIDS smoked more than half a pack of cigarettes daily compared to only 24 percent of male homosexuals who were symptom free. Marijuana and nitrite use also were significantly higher for AIDS cases than symptom-free controls.

Schechter et al. (1985) found homosexual males with antibodies to the AIDS virus were more apt to smoke (69 percent) than homosexual males with negative antibody status (62 percent). However, the difference in smoking rates was not significant. It is of interest, however, that these rates of smoking for both groups of homosexual males are considerably higher than the average smoking rates in the male population and, for some reason, higher for the male homosexuals with and without AIDS in the study of Newell et al.

Periodontal disease: MacGregor (1984) provided a brief review of 1) studies that indicated smoking to be associated with an increase in the severity of chronic inflammatory periodontal disease; 2) a smaller number of studies that did not find this

effect; and 3) several studies that consistently showed poorer oral hygiene in smokers compared to nonsmokers. MacGregor (1984) also provided data indicating more plaque before and after brushing of teeth in male and female smokers than male and female nonsmokers. Although no difference was found in frequency of subjects brushing their teeth between smokers and nonsmokers, male smokers brushed their teeth for a significantly shorter time than male nonsmokers, and a similar, though nonsignificant trend was found for females.

Depression: Frerichs et al. (1981) found smokers to have significantly more depression than nonsmokers as measured by the Center for Epidemiologic Studies-Depression Index. Kaplan et al. (1984) also found cigarette smoking associated with levels of depression. Depression was measured by the Beck Depression Inventory. Nonsmokers showed lowest depression scores with those who "never tried" lower than those nonsmokers who "have tried." Amount smoked was directly associated with depression scores. Salmons and Sims (1981) looked at individuals who were treated for neurosis and found they were much more apt to be smokers than individuals from a control group treated for varicose veins. Smokers in the neurotic group smoked nearly twice as much as the smokers in the varicose vein group. This increased rate of smoking in neurotics held for males and females in all ages and social classes.

Suicide: Given the association between smoking and depression described above, it is not surprising that smokers are more apt to commit suicide than nonsmokers. Niskanen, Tamminen, and Sakki (1978) studied female psychiatric inpatients. Drinking to the point of intoxication at least once a mo was reported by 88 percent of smokers and 21 percent of nonsmokers. Suicide attempts were reported by 40 percent of smokers and 14 percent of nonsmokers. This was true despite psychiatric disturbances being milder in the smokers, 26 percent having psychotic or borderline disorders as compared to 46 percent of the nonsmokers, and with schizophrenia four times as frequent in the latter. Suicide rates were high among schizophrenics and almost all of these men and women were smokers (Masterson and O'Shea 1984). Among the neurotics who were twice as likely to smoke as a control group (Salmons and Sims 1981), Sims (1984) later reported the rate of suicide was much higher than in the general population. However, no breakdown of suicide for smoking and nonsmoking neurotics was provided.

Even in normal populations, suicide is more frequent among smokers than nonsmokers. Paffenbarger, King, and Wing (1969) studied characteristics in youth that predisposed to suicide and accidental death in later life and found smoking in college was associated significantly with both the rates of suicide and accidental death in former Harvard University students.

Smokers comprised 43 percent of the men dying from suicide whereas they comprised only 33 percent of a randomly selected control group of students who lived at least as long as the suicides and did not die from suicide. A slightly higher percentage of smokers was found among men who died from accidents. Thomas (1976) also found medical students who had committed suicide were more apt to have been smokers than were their living and healthy classmates at the same age. Friberg et al. (1970, 1973) found the smoking member of twins who were discordant on smoking was more apt to die from suicide than the nonsmoker.

Work absenteeism differences between smokers and nonsmokers

Wilson (1973) analyzed data from the 1970 Health Interview Survey of noninstitutional US citizens. He considered illnesses of all kinds and reported smokers averaged 6.3 d work lost from work per yr versus 4.4 d on the average for nonsmokers. Former smokers averaged 5.2 d work lost per yr. Although the survey showed chronic respiratory conditions were more prevalent among smokers than nonsmokers, the amount of work loss associated with these diseases was estimated to be only five percent of the total time lost due to illness or injury. It may be that smokers are more willing than nonsmokers to report sickness when the time off is desired for other reasons (see Chapter 10: "Smoking, abuse of other substances, delinquency, and accidents").

A study by Athanasou (1979) of an industrial population found male smokers showed large differences from male nonsmokers in sickness-related work absence. Smokers averaged 13.8 d per yr of sickness absence compared to only 8.2 d per yr for nonsmokers. Female smokers and nonsmokers each averaged about 12 d per yr of sickness absence with no significant differences between the groups.

Holcomb and Meigs (1972) found a similar result when looking at factory workers in their fifties. Workers who had never smoked had a rate of 4.42 d work-loss per yr compared to 5.80 d work-loss per yr for smokers smoking less than a pack daily, 5.94 d work-loss per yr for smokers smoking a pack daily and 8.16 d work-loss per yr for smokers smoking more than a pack daily. Former cigarette smokers averaged 6.37 d work-loss per yr. Interestingly, current cigar and/or pipe smokers had only 3.22 d work-loss per yr. This latter result may reflect differences in work or socioeconomic position among pipe smokers, cigar smokers, and the other groups, possibly even a reluctance for managers to report or remember their illness-related absences when completing the questionnaire.

Athanasou (1975) reported research by Strnad, Fingerland, and Mericka (1969) which showed process workers in a machine plant had a much larger difference between smokers and nonsmokers in amount of absence due to illness (3.8 d) than occurred between nonsmokers and smokers who were technicians and clerks in the plant (0.6 d). In both cases smokers had more d of absence than nonsmokers, but the difference between the two worker groups supports a group difference explanation of the lower rate of absence for cigar smokers in the research of Holcomb and Meigs (1972). However, their study is not the first research to suggest pipe and cigar smoking are less hazardous than cigarette smoking (Bell and Laing 1969).

Military occupations show an association between smoking and absenteeism similar to the above results for civilians. Crowdy and Sowden (1975) studied respiratory ill-health among British soldiers and found hospital admissions for smokers were more than 30 percent higher than for nonsmokers. The duration of hospital stay did not differ between smokers and nonsmokers. However, Schmidt (1972) showed the number of d of illness with restriction to bed was 44 percent higher for smokers than nonsmokers in the German Federal Armed Forces. Unpublished data from the US Army Infantry Center at Fort Benning, Georgia, showed Infantry One Station Unit Training trainees who smoked had significantly more clinic visits than their nonsmoking counterparts (Blake, personal communication 1983).

Reports of research reviewed in this section largely have assumed the smoking relationship to absenteeism was related to higher levels of illness among smokers than nonsmokers. However, in a review of determinants of absenteeism in industry, Smith (1970) noted alcoholism and smoking were both strongly related to absenteeism. Given the strong association between smoking and alcoholism (see Chapter 10: "Smoking, abuse of other substances, delinquency, and driving accidents"), some of the association of smoking to absenteeism is undoubtedly an artifact of the smoking-alcoholism link. Research is needed to examine effects of smoking on absenteeism while controlling for alcohol consumption as well as consumption of other drugs.

A recent study of smoking and nonsmoking nurses by Parkes (1983) also found smokers to have significantly higher absence rates at work. She found the absence rate for nurses who reported smoking in stressful situations to be determined by their level of "affective distress." Since affective distress was higher for smokers, at least some of the relationship of smoking to absence rates of these nurses could instead be accounted for by their high levels of affective distress. Of course, long-term smoking itself may cause the affective distress (see Chapter 6: "Effects of smoking on arousal and ability to deal with stress, pain, and fear").

Conclusions and military implications

Research results from military populations and civilian populations of military age indicated significantly reduced disease of nearly all kinds and significantly lower absenteeism for nonsmokers compared to smokers. These facts provide strong reasons for reducing smoking in the military through increased selection of nonsmokers instead of smokers (when other factors are equal and a choice is possible) and through programs and policies that prevent the initiation of smoking and which discourage smoking among existing personnel.

Chapter 10

Smoking, abuse of other substances, delinquency, and driving accidents

Research over many years has frequently shown smoking is correlated positively with numerous undesirable traits and behaviors and the purpose of this chapter is to describe these findings. Only infrequently have the smokers turned out to be the "good guys" and these instances also are included.

To say that smoking causes the problems, such as drug abuse, which are described here, is usually inappropriate. Many of these smoker-nonsmoker differences seem to be related to differences between people at adolescence or early adulthood that lead some to smoke and some not to smoke. Some of these differences between eventual smokers and eventual nonsmokers exist very early in childhood. For example, Seltzer and Oechsli (1985) recently reported results indicating measures made long before children started to smoke showed significant differences between eventual smokers and eventual nonsmokers. These measures included Type A personality, extraversion, anger, "psychoticism," lower performance on intelligence tests, and lower performance on vocabulary tests. Oechsli and Seltzer (1984) showed family characteristics that existed at the birth of the children account for a significant amount of the variance related to smoking status of the children when they grew up. These variables included the mother's age and education and the father's education and occupation. Rantakallio (1983) obtained related results for family background variables including an association between smoking and being from a family with a large number of children, and between smoking and having a later birth position in the family.

Other evidence of "predestination" in smoking comes from studies showing lung function of boys who take up smoking is actually greater, on the average, than lung function of boys who do not (Tashkin *et al*. 1983). This correlation of superior lung function with smoking is more than a little ironic, since, as the authors point out, following several years of smoking, smokers' lung function will average less than that of nonsmokers. The existence of early predispositions to smoke also are the message of Kaprio *et al*. (1982) and Hannah, Hopper, and Mathews (1985), both of whom studied smoking in twins and showed genetic traits were highly important (along with environmental factors) in the development of smoking behavior. However, even when all of the early predispositions and family constellation variables related to smoking are considered, peer influences during critical early adolescent years appear to be the major determinant of whether or not a child actually will

take up smoking (e.g., Antonuccio and Lichtenstein 1980, Salomon et al. 1984).

What has been said above related to early determinants of cigarette smoking largely would apply to other forms of drug abuse as well. However, in the case of drug abuse, smoking frequently has been found to precede use of other substances (Smith and Fogg 1979). Interrupting the smoking link in the chain would have prevented some, perhaps much, subsequent drug abuse.

Smoking, alcohol use, and alcoholism

Dreher and Fraser (1967) found both male and female alcoholic outpatients were more apt to smoke than the general population. Only 7.3 percent of male alcoholics were nonsmokers compared to 37.1 percent of the general population. For females, these figures were 9.5 percent and 66.6 percent. They also found male and female alcoholic outpatients who smoke, smoked many more cigarettes than were smoked by smokers in the general population. Seventy-nine percent of male alcoholic smokers smoked over a pack daily compared to 32 percent of male smokers in the general population. Eighty-nine percent of female alcoholic smokers smoked more than a pack daily compared to 20 percent of female smokers in the general population. Although other psychiatric patients at that hospital smoked somewhat more than the general population, a later study showed they did not smoke nearly as as much as the alcoholics (Dreher and Fraser 1968).

Walton (1972) found 126 of 130 patients admitted for withdrawal from alcohol were smokers and 123 (97.6 percent) of these smoked a pack or more daily. Another group of 100 patients admitted to the same hospitals for reasons other than withdrawal from alcohol was comprised of 62 smokers and 38 nonsmokers with 46 (74 percent) of the smokers smoking a pack or more daily. Interestingly, Walton also found all four of the alcoholic patients who did not smoke were diagnosed as being schizophrenic.

Ayers, Ruff, and Templer (1976) also found alcoholic hospital patients smoked more than nonalcoholic psychiatric patients. Ninety percent of alcoholics smoked a pack or more daily versus 47 percent of the nonalcoholic psychiatric patients.

Maletzky and Klotter (1974) extended the results of Walton and Ayers, Ruff, and Templer to nonhospitalized alcoholics, including women alcoholics. Alcoholic groups had a higher proportion of smokers than controls (100 percent versus 65 percent

for male controls and versus 54 percent for females) and among smokers the alcoholics smoked a larger amount than the nonalcoholic smoking controls (48.7 versus 31.2 cigarettes daily).

Moody (1976) reported a similar result. He found 58 percent of nondrinkers also were nonsmokers. Among moderate drinkers, 24 percent were nonsmokers, while among problem drinkers, only 11 percent did not smoke. His sample consisted of patient volunteers at a southeastern medical center. Other research that indicates smoking is associated with moderate drinking and more strongly associated with heavy drinking was provided by Ferguson (1973). Borgatta and Evans (1968) studied entering university freshmen and found cigarette use was positively correlated with drinking beer and drinking hard liquor, as well as with the number of friends who "got drunk." Billings and Moos (1983) found heavy smokers were more likely to report drinking problems than nonsmokers, while light smokers did not differ from nonsmokers. As all of these various studies indicate, there is a striking direct relationship between smoking and problem drinking.

Part of the association between smoking and drinking may be accounted for by an increase of smoking caused by use of alcohol. Mintz et al. (1985) recently showed smoking increased during the drinking of alcohol for narcotics addicts participating in a methadone maintenance program. Significant increases occurred in the rate and amount of smoking with increases observed during drinking periods for 10 of 14 subjects. What is more, the four who did not show the increase were the heaviest smokers and a ceiling effect probably prevented any further increase in smoking following drinking. This coincided with a result for five alcoholics obtained by Griffiths, Bigelow, and Liebson (1976). However, generalization to the general population may be a problem for both of these samples.

Henningfield, Chait, and Griffiths (1984) studied the effects of ethanol on smoking alcoholic and nonalcoholic subjects. Only for the group of alcoholics did alcohol drinking produce significant increases in smoking. For the five nonalcoholic subjects, two showed substantial increases in smoking, two showed substantial decreases in smoking, and for the fifth, smoking remained unchanged. The sample of nonalcoholics was small, but there was a trend for smoking changes following ingestion of alcohol to be related directly to the subjects normal level of alcohol consumption.

However, Nil, Buzzi, and Bättig (1984) found the larger of two doses of alcohol did intensify cigarette smoking (larger puffs) compared to a control condition. Subjects were females who were regular smokers and who reported themselves to be in

good health and who were not alcoholics or drug abusers. Mello et al. (1980) also found increased smoking during drinking in normal social drinkers.. They followed occasional, moderate, and heavy smokers over 15 d of unrestricted alcohol availability and found increased smoking accompanied increased alcohol consumption for each type of smoker.

Although alcohol does appear to increase smoking, the major factor in the smoking-drinking relationship appears to be that youngsters who experiment with smoking also try drinking. Bloom and Greenwald (1984) found smoking and drinking were already significantly associated among fifth through seventh graders. Marijuana use also was associated with smoking and drinking. Rantakallio (1983) found smoking in 14-yr-olds was associated most strongly with alcohol use and "having been drunk" for both boys and girls in a study that examined associations between smoking and several dozen family and personal characteristics.

Smoking and other drug use

Bartol (1975) found smokers to be heavy users of coffee, amphetamines, and tranquilizers although no comparison was made with nonsmokers. Prendergast, Preble, and Tennant (1973) examined drug use and its relationship to cigarette and alcohol consumption among soldiers in West Germany and also among American high school students (military dependents) in West Germany. A strong association was found between cigarette consumption and drug use with users of drugs (marijuana, hashish, speed, LSD, etc.) nearly twice as apt to smoke as nonusers of drugs. When both alcohol consumption to the point of drunkenness and cigarette consumption were considered, drug use was found to occur among 75 percent of high school males who both abused alcohol and smoked, whereas only 19 percent of high school males who did not smoke or get drunk used drugs. Similar associations between smoking and heavy drinking and drug use also were reported for military males and high school females although actual percentages were not provided in the report.

Smith and Fogg (1979) found cigarette smoking was a very strong predictor of eventual use of drugs among US high school students. O'Donnell (1979) found use of cigarettes predicted marijuana use and use of other drugs, but that the relationship was small. He found marijuana use was a very strong predictor of use of other nonmedical drugs. This relative independence of marijuana use from cigarette use was described by O'Donnell as a fairly recent change. He noted that prior to 1970 it was highly unusual for marijuana use not to be preceded by cigarette smoking.

Hays, Stacy, and DiMatteo (1984) recently found three drug use measures (hard drug use, alcohol use, and cigarette use) were intercorrelated significantly, with the average of the three correlations among the three measures being .34 for high school males, .46 for high school females, .40 for college males, and .52 for college females. Separate correlations with cigarette use were not reported.

Von Knorring and Oreland (1985) studied 18-yr-olds in Sweden and found regular smokers were more prone to the abuse of alcohol, glue, cannabis, amphetamines, and morphine. Furthermore, they were much more apt to report alcohol-consumption-related blackouts and loss of control than nonsmokers, irregular smokers, or exsmokers.

Smoking and delinquency

A study of British 18-yr-olds showed 80.2 percent of delinquents smoked compared to 59.4 percent of nondelinquents (Knight, Osborn, and West 1977). In a study of Australian young people, Champion and Bell (1980) found tobacco use occurred in 82.3 percent of adolescent delinquents and this compared to 37.6 percent for nondelinquent students of comparable age. Alcohol use also was more likely to occur among delinquents (77.8 percent) than among nondelinquent youngsters (62.6 percent), but smoking much more reliably discriminated between the two groups.

Bell and Champion (1979) found 36.1 percent of 15 to 19-yr-olds in the general population of Australia who were low in antisocial deviance (had never or very infrequently been truant, committed traffic offenses, or ridden in stolen vehicles) smoked, versus 75.2 percent smokers among those who had frequently committed these minor offenses. An intermediate (moderate) group on antisocial deviance smoked at a rate of 56 percent. The proportions of low, moderate, and high antisocial deviants among this population of young Australians were .72, .15, and .13.

Reitsma-Street, Offord, and Finch (1985) compared antisocial boys and girls with same-sexed siblings who did not get into trouble. They found the problem children (a minimum of six instances of antisocial behavior) reported more frequent and heavier use of tobacco than their siblings who did not get into trouble. They also found these Canadian problem children who smoked, started smoking two yr earlier (10.8 yr of age for boys; 11.5 yr of age for girls) than smoking siblings who did not get into trouble (12.6 yr of age for boys; 13.7 yr of age for girls).

Bachman, Johnston, and O'Malley (1981) found truancy (self-reported frequency of cutting classes or skipping whole days of school) was a very strong predictor of cigarette and other drug use among American high school students. Only 25 percent of students reporting the lowest level of truancy smoked cigarettes compared to 60 percent of students reporting very high levels of truancy. Given this relationship of truancy to smoking, it is not surprising that high school grades also predicted smoking in this study. Approximately 18 percent of "A" students smoked cigarettes compared to 58 percent of students receiving "C-" or below. Simon and Primavera (1976) also found both high school and college grades of nonsmokers to be significantly higher than the grades of smokers. Rantakallio (1983) found a significant negative relationship between "ability in theoretical subjects" and smoking for both boys and girls. However, medical students in Yugoslavia showed no association between smoking status and academic success (Radovanovic et al. 1983). And smokers actually outperformed nonsmokers among undergraduates at the University of Reading in England (Warburton, Wesnes, and Revell 1984).

Truancy, poor family relations, poor academic performance, drinking, number of friends who smoked, and inability to resist peer pressure to smoke all were highly significantly associated with smoking in a study of tenth-graders in Jerusalem high schools (Salomon et al. 1984). Perceived popularity was significantly higher for smokers than nonsmokers and this drive to be popular may help explain the commencement of smoking and other drug use among adolescents.

Another manifestation of truancy in smokers was shown by Oldridge et al. (1978) who found smokers were less apt to comply than nonsmokers with an exercise program for men who had suffered myocardial infarction. Still another is the fact mentioned earlier (Chapter 5: "Effects of smoking on cognitive processes") that, in a study of smoker-nonsmoker differences in long-term retention (Mangan and Golding 1983), 20 of the 54 smokers failed to report for the retention session that occurred 1 mo following learning, despite prompting the day before, while none of the 15 nonsmokers failed to report and this was a highly significant difference.

Schofield (1969) found both male and female English teenagers who smoked were more sexually promiscuous than their nonsmoking peers. Twenty percent of nonsmoking boys were sexually experienced and 23 percent of nonsmoking girls. This compared to 50 percent of boys smoking ten or more cigarettes daily and 55 percent of girls smoking ten or more cigarettes daily who were "experienced." Borgatta and Evans (1968) found college students who smoked were much more apt to report their

friends engaged in petting and premarital sex than students who were nonsmokers. It is probable these behaviors applied to the smokers themselves as well, although such direct questions were not asked. Teenage girls who smoked in Bogalusa. Louisiana, were three times as likely to use contraceptives as their nonsmoking counterparts and this result held for both white and black girls in the study (Hunter, Webber, and Berenson 1980). Mayberry (1985a) showed smoking and genital herpes did not interact in the risk of cervical abnormality, although genital herpes virus antibodies were more common in smoking than nonsmoking women. Mayberry (personal communication, 1985b) found women in that study who smoked had first intercourse at an earlier age than women who did not and also found nonsmoking women were more than twice as likely to have only one life-time sex partner as were smoking women (68 percent versus 32 percent).

Zabin (1984) may have found the most striking relationship between smoking and promiscuity. She found age of first intercourse and level of cigarette smoking were associated strongly in young women visiting a contraceptive clinic. The relationship was striking particularly for whites. Among those having first intercourse at 12 or younger, 69 percent smoked more than one-half pack daily. This percentage of smokers declined linearly as the age of first intercourse increased. Only 14 percent of white girls having their first intercourse at age 18 or 19 smoked more than a half pack of cigarettes daily.

To classify promiscuity as delinquency may be unfair, and it may be even more unfair to include failure to use seat belts in this section on delinquent behavior. However, it is interesting that most studies that have investigated seat belt usage as a function of smoking have shown smokers are less apt to use seat belts than nonsmokers. Eiser, Sutton, and Wober (1979) found 30.5 percent of smokers, 48.4 percent of ex-smokers, and 56.8 percent of nonsmokers wore seat belts when riding in cars in a study of English adults. Williams (1973) found among US ninth grade boys and girls who smoked there was a significantly lower use of seat belts by smokers than by their peers who were nonsmokers. Cliff, Grout, and Machin (1982) found as cigarette consumption increased, the probability of using seat belts decreased, and in a later study Grout et al. (1983) reported 41 percent of nonsmokers "always wore seat belts" compared to only 18 percent for smokers. However, Helsing and Comstock (1977) found only a small association between smoking and seat belt usage with only the heaviest smokers differing from the nonsmokers and Eiser and Harding (1983) found no significant association between smoking and seat belt usage in a study of college and nursing students.

Driving accidents

The lack of use of seat belts among smokers is dangerous particularly since smokers are found to be more likely to have automobile accidents than nonsmokers. McGuire (1972) found a rate of 75 accidents per 100 drivers when drivers were heavy smokers, 59 accidents per 100 drivers when drivers were light smokers, and only 48 accidents per 100 drivers when drivers did not smoke. The subjects were young men who completed a questionnaire in their first week of Air Force basic training. Kraus et al. (1970) also studied young males and found 27 percent of the no-accident group had become regular smokers by age 16 compared to 39 percent of those males who had one or more accidents. Ashton et al. (1972) found some reaction times were elevated in smokers when they performed in a driving simulator relative to performance of nonsmokers (while others were reduced). These differences disappeared when smokers were not smoking. It is possible driving performance is impaired during smoking although reports by Heimstra typically have shown deprived smokers perform worse on driving simulators (see Chapter 4: "Effects of smoking on vigilance, rapid information processing, and divided attention").

Grout et al. (1983) found among people who had automobile accidents, the smokers were more likely to have their accidents at night (45 percent) than nonsmokers (20 percent). The corresponding figures for regular and occasional drinkers were 38 and 23 percent, and, given the correlation between even moderate drinking and smoking, it may be that alcohol use is a major factor in the relationship of smoking to driving accidents. Another factor in increased accidents at night for smokers suggested by Grout et al. is that smoke particles deposited on windshields may increase glare from oncoming headlights.

Strong evidence that drinking is a factor in the increased accident rates of smokers was provided by DiFranza and Winters (1985). They found 65 percent of Boston drivers arrested for drunk driving were cigarette smokers compared with 36 percent of drivers with no such arrests during the preceding yr. Compared with nonsmokers, smokers had a relative risk of 3.4 of being arrested for drunk driving.

Another possible explanation for this higher accident rate among smokers comes from the research of Williams (1973) who found impulsivity and chance-taking were positively correlated with smoking in teenagers while "harm-avoidance" was correlated with nonsmoking in this group. Similarly, Jacobs and Spilken (1971) found heavy smokers to be significantly higher on "defiant, impulsive, and danger-seeking" traits than nonsmokers. Simon and Primavera (1976) found smokers to rate them-

selves significantly lower on the adjectives "reliable" and "careful." Basic differences in personality may account for smokers' higher accident rate than the rate for nonsmokers. However, Schori and Jones (1977) did not find smokers (either smoking or deprived) to take more risks than nonsmokers in a passing situation on a driving simulator.

Nonsmoking and positive traits

Biersner, Gunderson, and Rahe (1972) found volunteers for strenuous underwater demolition training were much less apt to smoke than other Navy personnel and this would seem to indicate that at least some nonsmokers are "fighters."[1]

Hundleby (1985) compared tobacco and other drug use levels of high school students who had outstanding achievements in various fields such as academic performance, art, and music with tobacco and other drug use levels of high school students who were "nonoutstanding" performers. The outstanding performers were much more likely to abstain from tobacco (and other drugs) than the nonoutstanding performers.

Clarke, MacPherson, and Holmes (1982) found young adolescents who did not smoke were "internals", i.e., they believe they are able to determine their life outcomes through personal effort or ability. Smokers of comparable age were much more apt to be "externals," indicating they believe external factors largely control their lives.

Conclusions and military implications

Harm-avoidance and being careful may not be virtues in combat situations. Similarly, chance-taking and danger-seeking may not be vices. It is interesting to speculate whether smokers are not more apt to perform heroically in combat than nonsmokers. Along these lines, Heath (1958) reported Harvard students faced with wartime service in World War II were much more likely to volunteer for combat duty if they were smokers. Nonsmokers tended to select the Navy or to remain in civilian life. Case studies of the five heaviest smokers studied by Heath showed they typically had performed heroically in combat service careers.

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[1] Rates of smoking among enlisted and officer personnel in prestigious Army units, such as the Ranger battalions, should be investigated and compared to personnel in other units.

The results of Heath (1958) are from a period when smoking was much more widespread in the population and they applied only to a small number of Harvard University students. Research is needed to look at highly decorated combat veterans of more recent conflicts to determine if they were more apt to be smokers than their less heroic colleagues (see Chapter 12: "Needs for additional research on smoking and soldier performance"). One would hate to ban smokers or smoking in the military and discover, as a result, fighting effectiveness of units had diminished because the best "fighters" had left for occupations where smoking was permitted.

Undoubtedly, if the military somehow could restrict enlistments to nonsmokers, there would be far fewer discipline, alcoholism, and drug-abuse problems in the Army and other services. Of course, this is unrealistic and will become more so as changing demographics lead to sharp reductions in the number of youths available for military service in the next decade. If the military were to restrict driving to nonsmokers, there would undoubtedly be fewer vehicular accidents. If research were to show smokers had more accidents than nonsmokers while "driving" helicopters and other vehicles where losses from accidents are costly in terms of human life and dollars, it might strongly argue for nonsmoking drivers and pilots (see Chapter 12: "Needs for additional research on smoking and soldier performance").

Although enlisted acquisitions probably would not bear a screening out of smokers, except in MOSs viewed as particularly desirable, officer acquisitions might include smoking as a screening factor, or at least a negatively-weighted consideration, particularly for the prestigious officer training at the US Military Academy and the Branch Immaterial Officer Candidate Course, where there are many more applicants than can be accepted. Justification for this comes from the high probability that junior-officer leaders who are smokers will have larger numbers of smokers in their units and more smoking per smoking soldier, and, conversely, that nonsmoking leaders will have fewer smokers and less smoking among smokers in their units. Empirical data are needed to determine if the rate of soldier smoking is indeed related to military leader smoking (see Chapter 12: "Needs for additional research on smoking and soldier performance"), but this probably is the case for at least the following reasons. There is a strong influence of the presence of other smokers on whether or not people smoke (Antonuccio and Lichtenstein 1980, Glad and Adesso 1976). There is a potent effect of smoking status in older-sibling role models on whether or not a young person takes up smoking (Spielberger et al. 1983). And increased smoking was found in male students if the head teacher smoked cigarettes (Murray, Kiryluk, and Swan 1984). Since nonsmokers are less apt to

abuse drugs, to be delinquent, to be sick, to become pregnant, etc., this probable causal relationship between abstinence of smoking in leaders and relative abstinence in their soldiers should be explored and exploited if it does exist.

Chapter 11

Associations between smoking and other factors of potential relevance to soldier performance

Cerebral blood flow

Wennmalm (1982) reported the flow of blood to the brain increased by 25 percent immediately following smoking. However, in several studies where smokers were compared to nonsmokers, smokers showed lower levels of cerebral blood flow. Kubota et al. (1983) found a 12.5 percent reduction in smokers compared to nonsmokers. Rogers et al. (1984a) also found cerebral blood flow to be significantly lower in smokers who smoked more than a pack daily than in nonsmokers with the difference on the order of nine percent. Rogers et al. (1984b) showed responsiveness of cerebral blood flow to exposure to carbon dioxide and oxygen was reduced in smokers compared to nonsmokers. Exposure to oxygen normally reduces cerebral blood flow and such reductions were 24 percent lower for smokers who were not at risk for stroke and 34 percent for smokers who were at risk for stroke. Carbon dioxide exposure increases cerebral blood flow and such increases were 48 percent lower for smokers who were not at risk for stroke and 56 percent lower for smokers who were at risk for stroke.

The implications of these smoker-nonsmoker differences in cerebral blood flow for the increased stroke risk of smokers were discussed in Chapter 9: "Smoking-disease relationships: effects on productivity and absenteeism." However, cerebral blood flow is an important correlate of cognitive activity and shows variation across brain areas during different cognitive tasks (e.g., Roland and Friberg 1985). If smokers have a diminished capacity to muster needed blood to key areas of the brain, as the results of Rogers et al. (1984a, 1984b) suggest, this could account for results such as those of Elgerot (1976), who found poorer performance of smokers on difficult reasoning tasks including Raven's Progressive Matrices.

Given this decrement in cerebral blood flow as a result of habitual smoking, the question arises whether a change in smoking status will return cerebral blood flow to normal. Recently, Rogers et al. (1985) have shown abstention from cigarette smoking significantly increased cerebral blood flow, even after three or four decades of smoking. However, this increase was not to the levels of subjects without a history of cigarette smoking.

Slow wound healing and reduced reactive hyperemia

Mosely, Finseth, and Goody (1978) found injections of nicotine greatly delayed healing of wounds which were cut into the ears of rabbits. This experimental study had its origins when they observed a patient with a fingertip ulcer which did not heal until the patient terminated his heavy smoking. Possible mechanisms suggested for this retardation of the healing process included the vasoconstriction associated with nicotine which would reduce nutritional blood flow to the wound area. Smoking during recovery also has been found to be devastating for the results of surgery to reattach fingers (Wilson and Jones 1984).

Smoking also significantly attenuates reactive hyperemia which is the increase in blood flow to an organ that has had a temporarily reduced blood supply. This boost in blood flow helps to prevent tissue damage. There is both an immediate reduction of reactive hyperemia with smoking (Wennmalm 1979) and a difference between smokers and nonsmokers with reduced reactive hyperemia for smokers (Richardson 1985). Slow wound healing and reduced reactive hyperemia associated with smoking appear to have enormous implications for combat military operations with their heightened risk for injury.

In two recent papers research was reviewed on physiological effects of smoking in an effort to determine smoker risks following surgery and whether or not smoking should be terminated for some period prior to surgery (Jones 1985, Pearce and Jones 1984). It was concluded even 12 to 24 h of abstention would significantly reduce surgical risk with longer periods of abstention providing additional risk reduction.

Lung clearance

Cohen, Arai, and Brain (1979) used unique magnetic sensor technology to study long-term clearance of dust from the lungs. They found after 11 mo, smokers still retained 50 percent of the iron oxide (Fe_3O_4) dust they had inhaled into their lungs at the start of the experiment compared to only about 10 percent residual dust for nonsmokers. Vallyathan and Hahn (1985) compared whole left lungs of smokers and nonsmokers obtained at autopsy and found significantly greater concentrations of aluminum and silicon in the lungs of smokers. The concentration of these minerals in the lungs of smokers was associated significantly with the amount they had smoked during life.

Cohen, Arai, and Brain (1979) cite the decreased ability of smokers to clear dust particles from their lungs as a

possible factor in increased cancer among asbestos and uranium workers who smoked compared to their nonsmoking colleagues. Vallyathan and Hahn (1985) viewed both increased exposure to particles and decreased capacity for lung clearance as the basis for their result. High ability to clear dust particles from the lungs would appear to be very important for effective combat performance in dusty and smoky battlefields, as well.

Tobacco smoking effects on medical drugs

D'Arcy (1984) reviewed literature on the effects of tobacco smoking on the metabolism of a number of drugs. For the majority of drugs where smoking interactions had been examined, there was little problem. For insulin, propoxyphene, propranolol, and theophylline preparations there was some evidence of an interaction with tobacco smoking that could be of clinical importance. For example, smoking leads to increased dosage requirements for insulin. Vinarova, Vinar, and Kalvach (1984) found smokers needed higher doses of the neuroleptic drug chlorpromazine. The effect was explained on the basis of higher enzymatic activity in smokers which would accelerate the metabolism of the drug. Stimmel and Falloon (1983) found a patient had higher plasma levels of chlorpromazine and very different reactions to the drug when he quit smoking.

Research is needed to determine interactions of tobacco smoking with drugs, such as atropine, which will be used to counter nerve gas and other toxic agents on the battlefield. Presumably, the toxic agents themselves may have different effects on smokers and nonsmokers independent of countering drugs. Caffeine, for example, has been shown to be cleared nearly twice as fast in smokers as in nonsmokers (Parsons and Nelms 1978).

Varicocele incidence

A varicocele is a varicose vein which typically causes an enlarged mass in the left testicle. From 8 to 23 percent of all males suffer from this condition (Belker 1981, Handelsman et al. 1984). Varicoceles have been implicated in reduced fertility (Belker 1981), smaller left testicles (Handelsman et al. 1984), and increased height (Handelsman et al.). Although the research is controversial, some studies have reported diminished testosterone production in men with varicoceles, as well (Ando et al. 1983, Raboch and Starka 1971). Discomfort frequently is associated with the large testicular mass, particularly following strenuous activity. Klaiber, Broverman, and Vogel (1980) reported a much greater incidence of vari-

coceles among smokers (22 percent) than among nonsmokers (9.5 percent). However, Ducot, Mayaux, and Spira (1981) and Handelsman et al. (1984) did not find any difference between smokers and nonsmokers.

Research is needed to determine if this condition is related to smoking, and regardless of that association, research is needed to determine the extent to which a varicocele reduces testosterone production, alters growth patterns, and impairs performance of soldiers. Performance impairment could occur via hormonal mechanisms or simply for reasons of discomfort associated with enlargement of the scrotum. White et al. (1981) developed techniques making surgical correction of this condition possible on an outpatient basis.

Body size and weight

Seltzer (1959) reported heavy smokers were less apt to have a masculine physique than nonsmokers. Damon (1961) was not able to confirm this result, although smokers were found to be leaner than nonsmokers. Wack and Rodin (1982) found smokers generally have lower body weight than nonsmokers and that one frequent consequence of quitting smoking is weight gain. Hunger is one reliable correlate of smoking deprivation (West et al. 1984b).

Body sway

Uchida et al. (1980) found smoking of a single cigarette (12 deep inhalations at intervals of 15 s) produced a sharp increase in the amount of body sway that lasted for several min after smoking. Visual fixation on moving targets largely suppressed this smoking-induced increase in body movement. Closing the eyes caused it to return. Reflex eye movements of subjects during eye closure differed greatly between smoking and control conditions. During smoking, subjects produced short high-frequency eye movements instead of the slow large-amplitude movements that occurred prior to smoking. This reduction of subjects' eye drift following smoking may reflect the same process that caused changes of vestibular nystagmus with smoking reported by Tibbling and Henriksson (1968). Although Uchida et al. do not discuss practical consequences of these changes in posture, body sway, and eye movements with smoking, they may bear on the performance of soldiers in tracking and aiming tasks.

Sleep difficulty

Soldatos et al. (1980) measured sleep in a laboratory and found smokers took significantly longer to get to sleep than nonsmokers (43.7 min versus 29.8 min). Although coffee consumption was greater for smokers, this was shown not to be a factor in these sleep differences. A second experiment by these researchers looked at the effect of smoking withdrawal on sleep parameters. Smoking withdrawal led to a significant immediate increase in the amount of sleep, largely as a result of falling asleep much more quickly. This faster sleep occurred despite considerable daytime discomfort associated with the abrupt withdrawal from smoking.

Browman, Gujavarty, and Mitler (1984) found people with narcolepsy[1] reported less daytime sleep if they were smokers, but in a laboratory sleep experiment, narcoleptics who smoked actually fell asleep significantly more rapidly than nonsmoking narcoleptics. Narcoleptics who smoked slept an average of 40 min less at night than nonsmoking narcoleptics. This difference did not reach significance, but it may help explain the faster falling asleep during the day for smokers than for nonsmokers.

Palmer, Harrison, and Hiorns (1980) interviewed more than 800 people regarding their smoking and sleeping habits. Smokers slept less than nonsmokers and the amount of sleep was inversely related to the number of cigarettes smoked, particularly for males. Nonsmoking males averaged about 7.3 h sleep nightly. Males smoking more than 40 cigarettes daily averaged less than 6.5 h of sleep per night. Questions related to sleep quality showed some relationship between very heavy smoking and poor sleep quality in women. However, for men, there was no relationship between reported sleep quality and cigarette consumption, even among those who were very heavy smokers.

Hatsukami et al. (1984) found deprived smokers reported they awoke more frequently during the night and remained awake for longer periods than when they were not deprived of smoking materials. Hatsukami et al. noted the contrast between these self reports with the actual sleep data reported by Soldatos et al. (1980). A possible explanation of this difference which they suggested was that smokers, when deprived of tobacco, may have had more REM sleep which they perceived as not sleeping.

Bale and White (1982) administered a survey to women who were physical education or sports science students and found

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[1] Narcolepsy is a neurological disorder that leads to excessive sleeping during the day.

smokers, who constituted about 25 percent of the population, reported less h of sleep nightly on weekdays, more dreams, and poorer quality of sleep. Smokers also reported more headaches, more back injuries, more problems with nerves, more worrying thoughts, more depression, more bladder and menstrual difficulties, more weight loss, more use of unprescribed medicines, more alcohol use, and more alcohol abuse than nonsmokers with all differences significant at at least the .05 level.

It is not totally clear what the implications of this reduced amount of sleep for smokers are, or if and how the reduced sleep of smokers might affect soldiers in combat settings. Presumably, if nonsmokers get to sleep faster, this would enable them to get more sleep in extended combat operations where only "catnapping" is possible.

Left handedness

Harburg, Feldstein, and Papsdorf (1978) found left-handed people were significantly more likely to be smokers than right-handers. Sixty percent of the right-handed subjects in their sample were smokers compared to 78 percent of left-handed subjects. Among people who smoked, left-handers were found to smoke significantly more than right-handers. This association of smoking with handedness parallels findings of an association of birth stress (Bakan, Dibb, and Reed 1973) and alcoholism (Bakan 1973) with left handedness. A more recent study corroborated this difference between right-handers and left-handers for both smoking and drinking (Harburg 1981).

Passive smoking effects

Smoking not only influences the performance, mood, and health of the smoker, it also has effects on the health and attitudes of people around the smoker as well. Russell, Cole, and Brown (1973) found nonsmokers can develop levels of carboxyhemoglobin (COHb) comparable to those of light smokers just by breathing smoke-filled air in poorly ventilated rooms. The effects of COHb have been described in earlier sections and, although low levels generally have little measurable effect, it is possible some slight degradation of perception, endurance, and other performances can be expected as a result of passive smoking in very smoky environments.

Russell, West, and Jarvis (1985) recently showed passive smokers get only one-third of the nicotine a smoker gets from cigarette smoke. Nicotine apparently is associated with passive smoke particles that settle to the floor or do not otherwise find their way into the passive smoker's lungs. Any

nicotine-based benefit from smoking such as improved vigilance is not apt to be experienced by the passive smoker. However, the passive smoker gets a full passive-smoking dose of carbon monoxide and benefits from CO are largely nonexistent.

Health risks of passive smoking probably are real (Lefcoe et al. 1983, US Department of Health, Education, and Welfare 1979), but at least one pharmacologist generally disagrees (Aviado 1986). Passive smoking health risks certainly are dwarfed by the much greater health risk for the smoker himself (see Chapter 9: "Smoking-disease relationships: effects on productivity and absenteeism"). But it follows logically that if large exposure to cigarette smoke, as occurs in the moderate or heavy smoker is bad for health, lesser exposure through light smoking or passive smoking will be worse for health than no exposure at all. It is true the pattern of alcohol use and health is one where light-to-moderate use actually is better than none (Baum-Baicker 1985). For example, alcohol actually increases the amount of high-density lipoprotein cholesterol. However, this alcohol exposure benefit probably does not apply to cancers related to alcohol use which typically show increasing incidence as a function of dose. Given the major association between smoking and lung and other cancers, including direct associations between the rate of these cancers and the amount of smoking, benefits from small exposure to tobacco smoke probably do not exist like they do for alcohol.

Oborne (1983) found detrimental effects of passive smoking on an auditory detection task and on Raven's Progressive Matrices. Subjects performed these tasks twice, once with a confederate present who smoked to provide the tobacco smoke environment, and once with a confederate present who did not smoke. Sex of the confederate and order of smoking and nonsmoking trials influenced the results and Oborne did not view the decrement during passive smoking as an effect of the carbon monoxide, nicotine, or other component of the smoke inhaled by the subject. Oborne instead viewed subject annoyance with the smoking partner as playing a major role.

Other research has shown that passive smokers often are upset by the exposure to tobacco smoke (e.g., Zillman, Baron, and Tamborino 1981). Nonsmokers obviously are not receiving sufficient nicotine through their passive smoking to reduce aggressiveness as Cherek (1981) found reliably occurred for the active smoker. However, it is of interest that complaints about the effects of passive smoking tended to disappear if the passive smokers were highly involved in work tasks (Stone, Breidenbach, and Heimstra 1979).

Conclusions and military implications

Blood flow to the brain is critical for effective neural functioning and behavior. The diminution of such blood flow with long-term smoking may reduce cognitive performance and the increase in cerebral blood flow following smoking cessation may improve cognitive performance. The prospect of improved cognitive performance may be a strong motivator for smoking cessation for those people concerned about preserving and enhancing their cognitive performance.

The problems of smoking and the healing of wounds and for rapid restoration of blood to deprived tissues have tremendous implications for military personnel who are likely to receive battlefield injuries. Large military implications exist for the slow clearance of particles from the lungs by smokers given the heavy levels of smoke and dust on future battlefields. There also appear to be large military implications of the more rapid clearance of medical drugs by smokers which could interfere with drug treatment of illness or injury and interfere with drug pretreatments designed to protect against chemical agents.

Sleep will be a critical factor in continuous military operations (Department of the Army 1983) and the poorer sleep of the smoker may put him at a disadvantage compared to the nonsmoker and deprived smoker. However, more research is needed on the effects of tobacco use (or use of other forms of nicotine) in continuous operations. Increased body sway following smoking, as the earlier reported increased muscle tremor following smoking, could reduce marksmanship with rifles and other hand-held weapons.

Passive smoking may become an increased problem for performance of "sensitive" nonsmokers as more soldier operations take place in vans and other indoor settings and as more Army attention to the dangers of smoking increases nonsmoker sensitivity. Even if performance on some military tasks was shown to be improved following nicotine administration, passive smokers would not receive enough nicotine to share this benefit.

Chapter 12

Needs for additional research on smoking and soldier performance

Conflicting research results were described in the previous chapters and the recommendation frequently was made that additional data were needed to resolve these conflicts. In addition, in many instances data did not exist that related long-term and short-term effects of smoking to the intense stressors and unique tasks that characterize military operations. This chapter describes a number of key areas where additional research on smoking and soldier performance could provide major payoffs of improved soldier and unit performance, as well as payoffs in terms of increased knowledge about the effects of tobacco and nicotine on human performance. Some research needs which were discussed (e.g., the effects of smoking on duration of visual aftereffects and the smoking-eye-movement effects on scalp recordings of "brain" activity) primarily are of interest to physiological and psychological researchers and will not be redescribed.

Smoker, nonsmoker, and deprived smoker differences on military vigilance tasks

Smokers occasionally have been found to outperform nonsmokers on laboratory tasks requiring attention over long periods (Wesnes and Warburton 1978). Many military watch-keeping tasks in the field and before CRTs or other display devices in vans or other shelters, would appear to fit this vigilance-task category. Research is needed to determine if smokers have any advantage over nonsmokers in real-world vigilance tasks as well as laboratory vigilance tasks. If they do, other research is needed to determine if other sources of nicotine can provide the benefit since lighting and smoking cigarettes is not appropriate behavior for sentries, day or night. The effects of smoking deprivation on these military vigilance tasks also is of major interest. This research should compare soldiers who are nonsmokers, nondeprived smokers, and deprived smokers on their performances of military vigilance tasks.

Smoker, nonsmoker, and deprived smoker differences on rapid information processing tasks

A large number of operator tasks on new Army weapons systems involve monitoring of CRT displays and responding to rapidly changing displays on keyboards or other terminals. The

rapid information processing tasks studied by Wesnes (1985) and other researchers bear much resemblance to the tasks performed by operators of the Patriot air defense system or the Aquila remotely-piloted-vehicle. Research is needed to determine if performance on these critical operator tasks is different for operators who smoke and for operators who do not. For operators who are smokers, performance during conditions of smoking and during different periods of smoking withdrawal need to be compared.

Smoker, nonsmoker, and deprived smoker differences on complex military problem-solving tasks

Elgerot (1976) found an intelligence test, Raven's Progressive matrices, was performed better by smokers when they were deprived of cigarettes for several h. This result is contrary to many results for deprived smokers on vigilance tasks or rapid information processing tasks in which deprived smokers are at a disadvantage. The Elgerot result suggests that when decision-making tasks or other cognitive tasks are truly difficult, recent cigarette smoking is a hindrance. Unfortunately, little other relevant data exists and the Elgerot study needs to be replicated. Complex military problem-solving tasks should be used along with Raven's task to increase the validity of results from this important research.

Exercise duration and physical performance differences between smokers and nonsmokers

Smoker-nonsmoker differences in cardiovascular performance often did not appear in laboratory research if the duration of the physical exercise was short and the subjects were young. Two-mi runs, on the other hand, frequently showed differences between smokers and nonsmokers even when the subjects were less than 20 yr of age (Cooper, Gey, and Bottenberg 1968). One outcome of the jogging-running movement of the last decade has been to increase awareness that anyone has the capacity for prolonged aerobic activity given proper training. It is highly likely that the ability to run 10 mi translates into vastly improved performance in traditional infantry operations. Research is needed that compares smokers to nonsmokers on oxygen debt following cardiovascular work of varying durations including durations of more than a 2-mi run or its equivalent. Smokers should be broken down into, two or even three, categories based on the amount they smoke. Different age ranges such as 18-23, 24-30, and 30-40 should be tested to establish age effects along with smoking duration effects. Dependent variables would be the time required for the pulse to return to

preexercise levels and also the time required to run the different distances.

It is anticipated this research will show sharp deterioration in smoker performance compared to nonsmoker performance as the work duration increased. Similarly, smoker-nonsmoker differences would be expected to increase as age increased because of cumulative negative effects of smoking on the cardiovascular and pulmonary systems. Discussions with combat veterans indicate the cardiovascular demands of combat situations frequently greatly exceed the cardiovascular demands of any physical-fitness-test event. The results of this research could provide a basis for briefings, videotapes, or films informing soldiers and their leaders about the devastating effects that long-term smoking can have on their performance (and chances of survival) in combat situations.

Research on differences between smokers and nonsmokers on dark adaptation

The studies of Young and Erickson (1980) and Luria and McKay (1979b) indicated substantial differences favoring nonsmokers in the time required to detect dim targets following exposure to lights. Research is needed with a variety of dark adaptation tasks (e.g., different target distances, foveal targets, and peripheral targets) to determine if these large differences are real. If so, the results would provide some of the strongest bases for selecting nonsmokers for night fighting. Smokers in this research should be grouped by smoking experience with age-matched nonsmoker controls. Former smokers should be included to determine if any smoker-nonsmoker differences which might be identified are reversible. The Luria and McKay (1979a) improvement in smoker night vision over several h of abstention from smoking also should be examined in this research.

Research on the immediate effect of smoking on dark adaptation

In no area (except perhaps the effects of smoking on testosterone production) were more contradictory results reported here than in the research on the immediate effects of smoking on rate of dark adaptation and final night vision levels. Some studies found decrements (Sheard 1946), others found improvement (Troemel, Davis, and Hendley 1951). The general belief is smoking is detrimental to night vision. However, it could be that lighting of cigarettes caused additional light adaptation in those studies where poorer dark adaptation following smoking was the finding. Night vision is critical for many military missions, especially during continu-

ous combat operations. It is important to determine whether or not tobacco use is detrimental to this performance, or whether or not nicotine (preferably administered by nonsmoking methods) might actually facilitate vision in low light levels as some studies suggest. Since smoker-nonsmoker differences in night vision and dark adaptation were controversial and since nonsmokers also might benefit from nicotine administration in tablet, gum, or aerosol form, this research on the immediate effects of smoking or other forms of nicotine administration should include a nonsmoker control group.

Smoking and changes in ocular accommodation and convergence

In darkness and in visual situations where there is little or no contour, the eyes tend to adopt a "resting" position of about two-thirds of a m (Leibowitz and Owens 1978). This applies to the convergence of the eyes and also to the state of ocular accommodation of each eye. No research has been conducted on the effects of smoking or nicotine on the resting position of accommodation and convergence. As mentioned in Chapter 3: "Effects of smoking on perceptual processes," some of the contradictory findings in regard to smoking and dark adaptation could have resulted if smoking caused changes in the focus of the eyes that made dark adaptation test targets less visible for smokers than for nonsmokers (or conversely, made them more visible for smokers than nonsmokers in those studies that showed smoking to facilitate dark adaptation). The research by Tibbling (1969) and Uchida et al (1980) showed the influence of smoking on extraocular musculature and these results augur for effects on the intraocular ciliary muscle that changes the shape of the crystalline lens, as well. Research is needed to measure the effects of smoking and smoking deprivation on the resting states of accommodation and convergence. This research would use accommodation-measurement techniques such as the laser scintillation optometer (Leibowitz and Owens, 1978). One possible outcome would be that smoking causes pilot visual performance to be attenuated when looking through windscreens for distant aircraft.[1] Alternatively, smoking deprivation may be a potential threat to a pilot's control of visual accommodation.

[1] Test pilot Chuck Yeager claimed his success as a fighter pilot was largely related to his ability to control his accommodation and to focus his eyes at great distances even when there were no objects at those distances.

Effects of smoking on "flinching" and other factors in marksmanship training

Smoking reduces the magnitude of large-muscle reflex activity (Domino and von Baumgarten 1969) and one expected effect of smoking would be to reduce the magnitude of large muscle movements such as flinching while shooting. Another expected effect of smoking would be to reduce the ability to hold the weapon steadily due to sharply increased muscular tremor (Lippold, Williams, and Wilson 1980). Ironically, smoking thus could potentially improve the performance of the bad shooter while hurting the performance of the good shooter. Given the wide range of perceptual and motor systems involved in rifle marksmanship, other effects of smoking on marksmanship than these also might occur. For example, the speeding of some mental and perceptual processes by nicotine and smoking (Wesnes 1985) suggests a possible improvement of performance following smoking in situations where numerous targets appear for brief periods and rapid pointing and shooting of the weapon is required. In addition, the pronounced increase in front-to-back body sway found by Uchida et al. (1980) suggests smoking would have a substantial effect on a task such as off-hand rifle shooting or tracking of moving targets with a hand-held weapon or other tracking device. Research is needed to determine whether these body-sway changes with smoking do have any detrimental effect on such real-world tasks. The time course of any such effects during and following smoking also would be of interest. Empirical data are needed that look at the effects of smoking on shooting as a function of time since smoking. Other research is needed which compares smokers, nonsmokers, and deprived smokers on different shooting tasks.

Effects of smoking on arousal in stressful training settings

Many indexes of physiological arousal typically show increases with smoking (Gilbert 1979). However, most studies of the effects of smoking on physiological arousal use subjects who are in relatively nonstressful situations. Research is needed to assess the effects of smoking on physiological and subjective arousal when the subjects are highly aroused for reasons other than heavy physical work. Airborne trainees awaiting their initial airplane jump, constitute such a highly aroused population. Smoking of cigarettes with different levels of nicotine and administration of nicotine by nonsmoking methods, such as tablets or aerosols, may reduce physiological arousal in these situations. Even heart rate, which nearly always is elevated by smoking, might decline in this highly stressful situation which produces highly elevated heart rates in most trainees (Dyer and Burke, unpublished study). If so,

this finding would help reduce Nesbitt's Paradox by showing emotion-calming and reduced physiological arousal do occur together. Performance of nondeprived smokers, deprived smokers and nonsmokers should be compared in this research.

Effects of smoking and amount of smoking on success in stressful training

Nesbitt's Paradox (Schachter 1973) provides contradictory predictions of the effect of smoking on success in stressful training such as Airborne and Ranger training. If the perceived calming of the emotions were the key result of smoking, this could reduce stress and facilitate performance. If the major effect of smoking were only to further boost physiological arousal, smoking could impair performance. Many of the stressors in Airborne and Ranger training involve heavy physical work and smoking would be expected to reduce the capacity of trainees to perform adequately. Research is needed to determine the differences in training attrition and training performance for smokers and nonsmokers in different Army training situations. Smokers need to be categorized into light, medium, and heavy user smoking categories to determine if the amount of smoking predicts training success. To the extent possible, the specific reasons for smoking's negative and/or positive effects need to be determined along with effects on training outcomes.

Smoker-nonsmoker differences in drug abuse and delinquency in Army settings

The potent associations between smoking and drug abuse and between smoking and delinquency that repeatedly have been shown in civilian populations argue that military problems related to drug abuse and delinquency would also be associated with smoking. It is important to conduct research that accurately describe these probable smoking-delinquency and smoking-drug-abuse relationships in military populations. Presumably, annual surveys of soldiers could include questions on smoking behavior and these data on smoking incidence and smoking frequency compared between delinquent and nondelinquent groups, soldiers who abuse drugs and soldiers who do not, unmarried soldiers who became pregnant and those who do not, etc. Alcohol use should be measured in this research since many of the differences between smokers and nonsmokers may reflect the increased use of alcohol of the smokers (Hays, Stacy, and DiMatteo 1984).

Effects of leader smoking behavior on amount of smoking in the unit

The strong effect the presence of other smokers has on smoking (Antonuccio and Lichtenstein 1980) and the importance of the smoking behavior of older siblings in determining whether people take up smoking (Spielberger et al. 1983), both suggest that smoking by leaders in Army units would increase smoking among troops in the unit. Research is needed that relates amount of smoking in the unit to the smoking behavior of the leaders. The expectation is that relative to nonsmoking leaders, leaders who smoke will have more smokers in their units and also more smoking by these smokers. Time of leader assignment to the unit, level of leadership, and numerous other factors need to be taken into consideration in such research, but it is anticipated such a study is feasible. Given the many health problems and other problems associated with smoking, knowledge that smoking in units can be influenced by leader smoking behavior would be useful. For example, it could influence leadership training and leader selection.

Differences between smokers, nonsmokers and deprived smokers as a function of time in MOPP

The gas mask and other protective clothing that constitute the Mission-Oriented-Protective Posture (MOPP), provide an exceedingly stressful work environment for soldiers that normally only can be tolerated in training settings for a few h. Smoking is impossible in MOPP and withdrawal symptoms would be expected to add an additional MOPP burden for smokers that would not exist for nonsmokers. Breathing through the gas mask filter also may be more difficult for smokers who eventually show impairment of lung function compared to nonsmokers (Tashkin et al. 1983). These factors would predict large differences between smokers and nonsmokers in their performance in MOPP operations. Research is needed to confirm or repudiate this prediction.

Effects of smoking on performance on the Army Physical Readiness Test

Nonsmokers outperform smokers on physical fitness tests, particularly on those events that require endurance. Rode and Shephard (1971) showed brief abstention from smoking improved performance. Other research has shown immediate decrements in performance with recent smoking (Hirsch et al. 1985). Research is needed that compares smokers with different levels of tobacco deprivation (0, 1, 2, 4, 8, 16, and 32 h) to determine the nature and duration of effects of smoking on physical

performance. It is unrealistic to expect most soldiers who are habitual smokers to give up tobacco. If, as expected, results of this research showed relatively brief smoking deprivation periods enhanced physical performance, this would provide a basis for short-term deprivation prior to physically demanding Airborne testing, APRT-testing, and of most importance, the physically demanding test of combat.

Effects of smoking on optokinetic nystagmus and detection of targets from moving vehicles.

Tibbling (1969) showed vestibular nystagmus changed dramatically with the frequency of eye movements doubling and the amplitude of the slow "tracking" phase cut by one-half. Uchida et al. (1980) found eye movements under closed lids also decreased in amplitude and increased in frequency. Although both of these eye movement changes occurred with an absence of visual input, they suggest a possible change in optokinetic nystagmus, the eye movements associated with eyes-open viewing of vertical contours moving rapidly in a horizontal direction before the eyes. No research was found on the effects of smoking on optokinetic nystagmus, but if smoking-induced changes in optokinetic nystagmus occur, it may be that tracking of moving targets also changes under the influence of smoking. Such moving targets might be the result of target movement, movement of the soldier in a rapid vehicle, or both. Basic research is needed on the effects of smoking (and smoking deprivation) on optokinetic nystagmus. Given that smoking effects occur and are nontrivial, applied research should follow on eye-movement behavior and visual performance in aircraft and fast-moving land vehicles where soldiers are searching for military targets. A nonsmoker control group should be included in both the basic research and any subsequent applied research.

Effects of sleep deprivation on smokers and nonsmokers

The research that has shown smokers to require a substantially longer period to fall asleep (Soldatos et al. 1980) could have major implications for soldiers who are operating on reduced sleep schedules. If nonsmokers continue to fall asleep faster than smokers under such conditions of sleep deprivation, they probably would show a less detrimental effect from sleep loss, simply because they were experiencing less sleep loss. However, the stimulation associated with nicotine from smoking might allow smokers to function during sleep-deprivation in tasks which led their nonsmoking colleagues to fall asleep. Studies of sleep loss and sleep rationing should include smoking as an independent variable, comparing smokers and

nonsmokers, comparing heavy smokers with light smokers, and assessing the acute effects of smoking and smoking deprivation.

Smoker-nonsmoker differences in incidence of military land vehicle and aircraft accidents

Given the reliable association between smoking and automobile accidents in nonmilitary settings (McGuire 1972), one would expect that military personnel who smoke also would have more accidents than their nonsmoking counterparts. Given that every soldier does not need to be a driver, at least in peacetime, research is needed to determine if any worthwhile advantage would be gained from not granting military drivers' licenses to smokers. Similar research is needed to look at aircraft accidents as a function of smoking status of the pilot. Unfortunately, if this hasn't already occurred, it is planned to drop smoking status of the pilot from future surveys of military aviation accidents. What is needed instead is to begin to collect smoking status of all pilots to allow valid comparisons of smoking status for those who have accidents and those who don't.

Smoking and incidence of varicocele in soldiers

Klaiber, Broverman, and Vogel (1980) found a strong association between smoking and the presence of a varicocele. Varicoceles are uncomfortable if they are large and, as a result, they probably impair physical performance of soldiers who have them. Since varicoceles may reduce production of testosterone, the condition may have additional negative consequences for soldier performance, aggressiveness, and leadership. Research is needed on the incidence of varicoceles among soldiers and relating this condition to smoking, hormone production, task performance, and even body structure.

Smoker, nonsmoker, and deprived smoker differences in tolerance of food and water deprivation

Legend has it the time the water was slow arriving at the bivouac site during basic training was planned as a way to teach soldiers to cope with the stress of water deprivation. Undoubtedly, the double deprivation associated with not smoking and no water or no smoking and no food, would be a larger stressor than deprivation of water or food alone. However, this needs confirmation in a research setting. Of more interest is the question of whether the nondeprived smoker might tolerate the stressors of water or food deprivation better than the nonsmoker. Nondeprived smokers appear to

tolerate the stressor of prolonged boring work better than nonsmokers in some instances (Heimstra, Bancroft, and DeKock 1967). They also seem to tolerate pain better than nondeprived smokers (Nesbitt 1973). An experiment that compared the reactions of nonsmokers, deprived smokers, and nondeprived smokers to food or water deprivation would answer both sets of questions. Subjects' rights must be carefully weighed in any decision to conduct such research.

Smoker, nonsmoker, and deprived-smoker differences in performance in combat situations

The stressors of combat far exceed any which can be legitimately imposed in experimental research. Smoking may serve a unique function in allowing men to tolerate these stressors. Another less likely possibility is smoking may actually add to these stressors and impair performance. The third alternative is smoking will make no difference. Memories of combat veterans may shed at least some light on these important questions. Structured interviews conducted with combat veterans who were smokers could obtain at least subjective reactions to the effects of smoking during crises. Former combat leaders may be able to give another perspective on the effect smoking had on troop performance in stressful combat situations. Obviously, some firefights allow no more opportunity for smoking than occurs for players in a football game. (Chewing tobacco and snuff use may occur in these settings, however, and the effects of nicotine from these tobacco forms are somewhat similar to the effects of nicotine derived from smoking tobacco. The point is even for an infantry engagement, effects of tobacco use need to be considered.) Other situations such as the "desk" work associated with field-artillery computations probably allow unrestrained smoking. Questions to combat veterans regarding amount of smoking in these situations and smoking payoffs and/or problems (e.g., position disclosure) could provide invaluable information both for our knowledge of the effects of smoking on tolerance of truly major stressors, but also information of value to commanders who could control the smoking of their troops to maximize soldier performance. Veteran combat leaders who smoke and veteran combat leaders who do not might have different perspectives on tobacco's effects and both groups should be included in the research. This study also could include a comparison of indices of combat performance (medals, citations, etc.) between smokers and nonsmokers since there is some indication smokers may have performed more heroically than nonsmokers in World War II (Heath 1958).

Research on nicotine pills and aerosols as tobacco substitutes and performance enhancers

Research is needed to evaluate the performance, addiction and health implications of new means for providing nicotine (Russell et al. 1983). Typically, these nonsmoking sources of nicotine have been used to provide nicotine during early periods of smoking cessation clinics to reduce withdrawal symptoms, but with only limited success. The nicotine aerosols appear to hold more promise than nicotine tablets or nicotine gum as smoking substitutes since they deliver nicotine to the body and brain in a dose somewhat comparable to the large rapid dose obtained from inhaling burning tobacco. The use of gum, tablets, or aerosols certainly would not give away a soldier's position when used and the addicted soldier whose performance would deteriorate if he were deprived of smoking might benefit greatly from their use. These alternative means to provide nicotine also have potential for abuse, however, and the research should be designed to evaluate this possibility.

Resolution of contradictory results related to smoking and cold injury

Some studies of frostbite showed an increased incidence among smokers (Sumner, Criblez, and Doolittle 1974) and others did not (Schuman 1953). Heavy smokers had fewer frostbite problems than light smokers in another study (Miller and Bjornson 1962). More data are needed to resolve the questions raised by the apparent contradictions in these research results. Different areas of the body which receive frostbite injury should be considered in this needed research, given the results of Suter, Buzzi, and Bättig (1983), who found vasoconstrictive responses to nicotine were "considerable with the finger recordings, modest with the foot recordings, and absent with the forehead and the ear recordings."

Effects of smoking on testosterone production

Male hormones may be critical to effective combat performance of soldiers and their leaders. The highly contradictory results related to differences between smokers and nonsmokers in levels of testosterone (Shaarawy and Mahmoud 1982, Deslypere and Vermeulen 1984) indicate a need for additional research to identify the circumstances when testosterone levels are positively and negatively associated with long-term smoking or to determine which relationship holds if some of the previous research is invalid.

Research on the effects of smoking on clearance of atropine and other drugs

Research is needed to determine tobacco smoking interactions with drugs such as atropine which will be used to counter nerve gas and other toxic agents on the battlefield. The toxic agents themselves may have different effects on smokers and nonsmokers independent of countering drugs and research on drug effects should compare smokers and nonsmokers, and also assess acute effects of smoking. Caffeine, for example, has been shown to be cleared nearly twice as fast in smokers as in nonsmokers (Parsons and Nelms 1978).

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